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Volume 18, Number 4

OCTOBER 1955

Postgraduate Medicine

Official Journal
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See SAUNDERS Advertisement on pages A-14 and A-15

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Postgraduate Medicine

Volume 18 · Number 4

OCTOBER · 1955

OFFICIAL JOURNAL OF THE INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION

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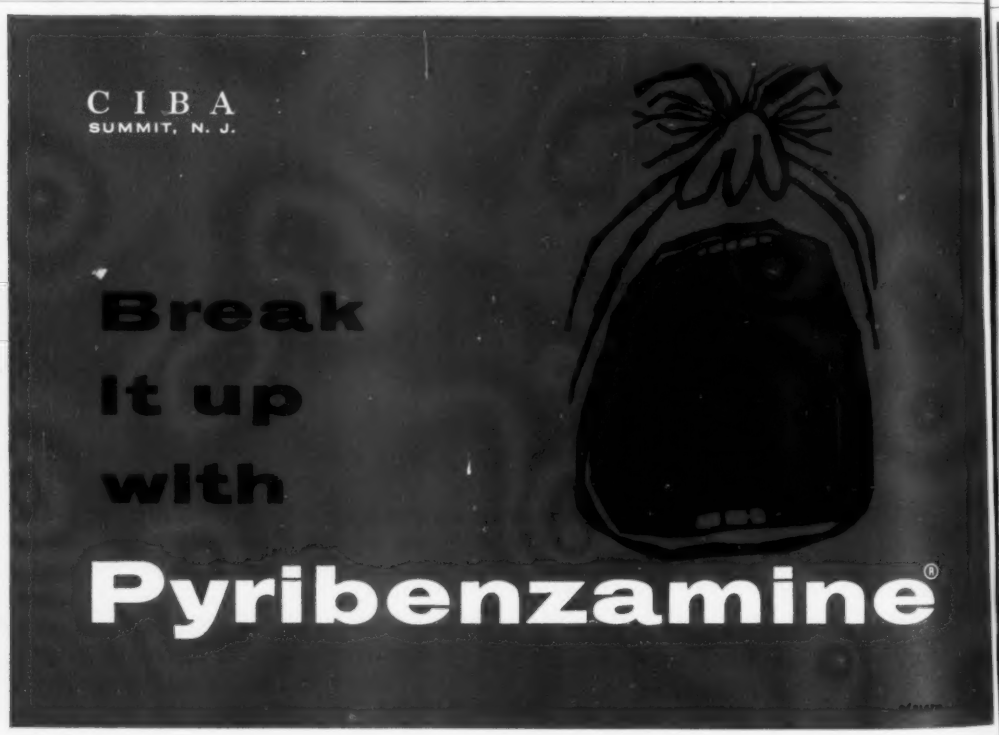

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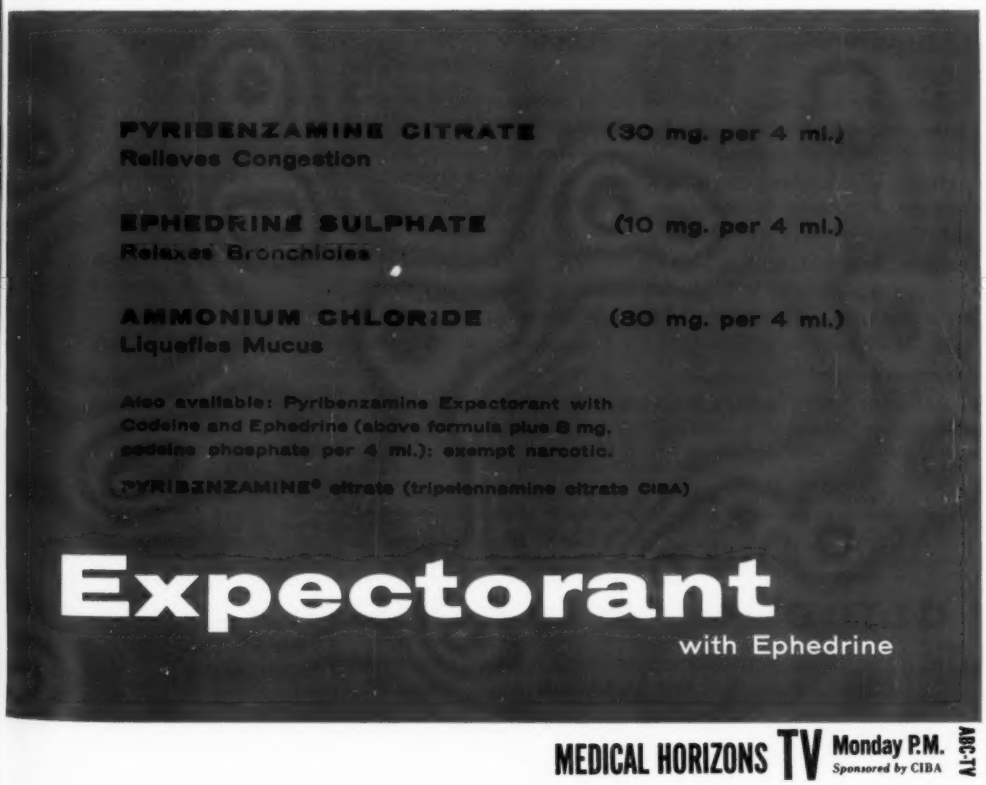
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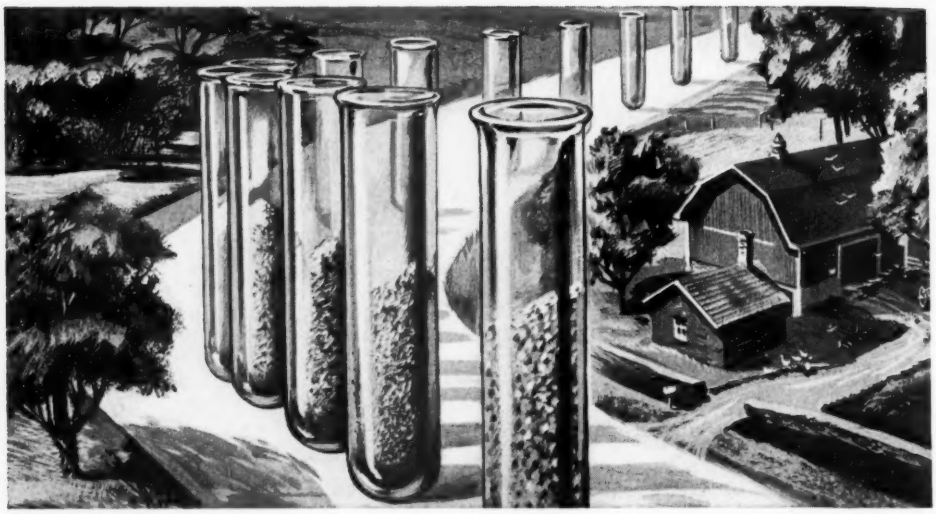

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Administration: "RG" Lecithin is presented in palatable granules which may be taken plain, in milk, in orange juice or other citrus juices, or sprinkled on cereal.

Literature available on request.

Bibliography: 1. Adlersberg, D., and Sobotka, H.: J. Nutrition *25*:255 (March) 1943. • 2. Adlersberg, D., and others: Gastroenterology *10*:822 (May) 1948. • 3. Adlersberg, D.: New York J. Med. *44*:606 (March 15) 1944. • 4. Adlersberg, D., and others: Am. J. Digest. Dis. *16*:333 (Sept.) 1949. • 5. Augur, V.; Rollman, H. S., and Deuel, H. J., Jr.: J. Nutrition *33*:177 (Feb.) 1947. • 6. Gross, P., and Kesten, M. B.: New York J. Med. *50*:2683 (Nov. 15) 1950. • 7. Schettler, G.: Klin. Wchnschr. *30*:627 (July) 1952. • 8. Dietrich, H. W.: South. M. J. *43*:743 (Aug.) 1950.

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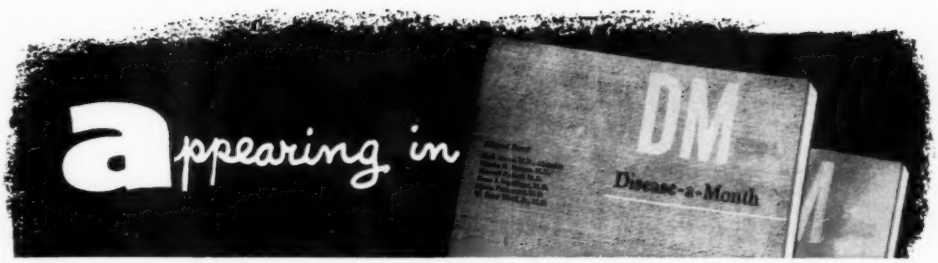

October issue of

Disease-a-Month Series

Chronic and Recurrent Diarrhea

BY THOMAS P. ALMY, M.D.

Associate Professor of Medicine, Cornell University

Disease-a-Month Series (DM for short) is only one year old, but how it is thriving! Thousands from almost every field in medicine, but especially those in general practice and internal medicine, have subscribed for this unique new series of monthly clinical monographs on current medical problems.

DM was conceived out of recognition of a growing threat to best medical practice; namely, inability of most doctors to find time for essential professional reading. Study of this problem indicated, however, that, regardless of how busy a man was, he would and could find time to read regularly a single and original 30 to 40-page monthly monograph—a concise, specific discussion of high authority, devoted to latest clinical management of a frequent and vital practice problem.

Thus DM was born; thus is it flourishing—concisely written; compact, pocket-size in format; practical and specific on up-to-date diagnostic and therapeutic procedure; custom-tailored throughout to the needs of the busiest practitioner.

Dr. Almy's monograph on Chronic and Recurrent Diarrhea in the October issue is typical of what you get in DM, and how. Below is a list of some of the future DM's and the authorities writing them.

DM is new and unique, but not just for the sake of being different. DM fulfills an important function; does it well. *Try it and see!*

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Rheumatoid Arthritis, *by Charles Ragan and Arthur I. Snyder*

Diabetes Mellitus, *by Max Miller*

Glomerulonephritis, *by David P. Earle*

Convulsive Seizures, *by H. Houston Merritt and Sidney Carter*

Cerebral Vascular Accidents, *by Joseph M. Foley*

Chronic Nontuberculous Bronchopulmonary Suppuration, *by Donald S. King*

Obesity, *by William Parson*

Renal Failure, *by Arnold S. Relman*

Antibiotics, *by Harry F. Dowling*

Treatment of Tuberculosis, *by Robert H. Ebert*

DM yb

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Your Questions answered

Readers may send questions from their own practice or other medical problems which will be answered by qualified consultants. Replies are forwarded by mail immediately and selected questions and answers are published each month in this section.

• RACE

Q. Every time I run into the question *Race* on an insurance examination application or some other form, I am at a loss as to the answer. I would appreciate your referring this question to your consultant in anthropology for the proper answers.

M.D.—Wisconsin

A. Scientifically we classify Man as follows:

I. Genus: Homo
  A. Species: sapiens
    1. Subspecies
      Caucasoideus (Europaeus)
      Negroideus (Africanus)
      Mongoloideus (Asiaticus)
      a. Sub-subspecies or geographical "races" in stricter sense

Politically or legally it is generally the subspecies that is required as the answer on certain examination forms. In the United States, I think the examining physician should fill in *White* for white Americans, *Negro* for colored Americans, *American Indian* for Amerinds. "Whites" are Caucasoids; Negroes are Negroids; Amerinds are Mongoloids.

As a rule if any further breakdown is needed, for whites the nationality, or for Europeans the country of origin may be given, at least back to great-grandparents.

In cases of race mixture the law follows the "one-drop-of-blood" rule. A Caucasoid-Negroid cross is *Negroid;* a Caucasoid-Mongoloid cross is *Mongoloid;* a Negroid-Mongoloid cross is, if the Indian or tribe is a ward of the federal government, *Mongoloid.* In this latter category, if the mixture is known, it is best to write *Indian* and *Negro.*

• SHOCK FOLLOWING MYOCARDIAL INFARCTION

Q. What problems arise in the use of an oxygen tent for a patient with acute myocardial infarction in shock? Why are some patients uncomfortable, feel very warm and perspire excessively while in a tent? Is this due to carbon dioxide accumulation? Is there an advantage in having the tent at 60° F. in order to reduce the metabolic needs of the patient? Would such a low temperature cause a further stress stimulus, resulting in a greater increase in endogenous cortisone and if so, what effects might this have on the patient in shock due to the infarction? Would the hypotensive and peripheral vasodilation effect of chlorpromazine contraindicate its use in such a patient? Further, how does a LEVOPHED® drug effect vasoconstriction of the coronary arteries, since Levophed causes generalized vasoconstriction?

M.D.—Ontario

A. The problems arising in the use of an oxygen tent for a patient with acute myocardial infarction in shock are chiefly those of maintaining a high oxygen intake while the intensive treatment which is so often necessary for the

(Continued on page A-26)

the drug of choice

. . . as a tranquilizing agent in anxiety and tension states

. . . in hypertension

RAUDIXIN

Squibb Whole Root Rauwolfia

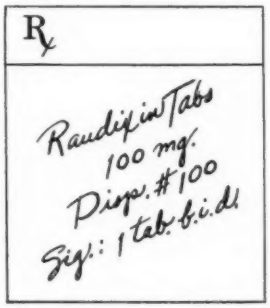
As a tranquilizing agent in office practice, Raudixin produces a calming effect, usually free of lethargy and hangover and without the loss of alertness often associated with barbiturate sedation. It does not significantly lower the blood pressure of normotensive patients.

In hypertension, Raudixin produces a gradual, sustained lowering of blood pressure. In addition, its mild bradycardic effect helps reduce the work load of the heart.

- **Less likely to produce depression**
- **Less likely to produce Parkinson-like symptoms**
- **Causes no liver dysfunction**
- **No serial blood counts necessary during maintenance therapy**

- Raudixin is not habit-forming; the hazard of overdosage is virtually absent. Tolerance and cumulation have not been reported.
- Raudixin supplies the *total* activity of the whole rauwolfia root, accurately standardized by a rigorous series of test methods. The total activity of Raudixin is not accounted for by its reserpine content alone.

Supply: 50 mg. and 100 mg. tablets, bottles of 100 and 1000.



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shock state is being carried out. An occasional claustrophobic patient will not tolerate the tent or mask, and the severe agitation secondary to its use may do more harm than the benefit derived from the oxygen.

A patient who perspires excessively after an acute infarction will do so whether he is in an oxygen tent or not. This excessive perspiration is due partly to reflex autonomic hyperactivity and partly to the frequent profound mental stimulation resulting from the anxiety produced by the pain and trauma of the infarct itself. During certain stages after the infarction, such a patient presents many of the features found in hyperthyroidism, particularly the great stimulation and heat intolerance which he so commonly exhibits. In this way it is comparable to the "fight or flight" situation as described by the endocrinologists.

The purpose of a cool oxygen tent in situations like this is to make the patient comfortable rather than to produce hypometabolism. The temperature in the tent should be adjusted to the needs of the patient. If it is done this way, it should have no deleterious effect on a patient in shock due to the infarction.

Administration of chlorpromazine would allow considerably smaller doses of morphine to be used in controlling cardiac pain, but since one cannot always tell which patient is going to experience shock after his infarction, it would seem wisest to use enough narcotic drug to alleviate the pain rather than to rely on the potentiating effect of the chlorpromazine. Chlorpromazine could also inadvertently result in an overdose of the narcotic and thus contribute to the shock. If the patient has undue anxiety during his convalescence and if it is not controlled by ordinary means, treatment with chlorpromazine may be tried. This should seldom be necessary and should be reserved for those patients with more profound mental disturbances. The effect of chlorpromazine on the blood pressure should rarely be a problem but should be watched carefully during the first few days of the use of the drug.

Clinical reports indicate that norepinephrine has a favorable effect on shock due to myocardial infarction. Its effect is similar to but less than that of epinephrine in dilating the coronary arterial tree. Since epinephrine increases the blood pressure by increasing cardiac output, whereas norepinephrine increases the blood pressure by

(Continued on page A-30)

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Rapid, Prolonged Antacid Relief ... Balanced antacids — no laxation — no constipation

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Anti-enzyme Action ... Necrotic pepsin and lysozyme action checked

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Each 10 cc. of KOLANTYL Gel or each KOLANTYL tablet contains:

Bentyl Hydrochloride.....5 mg.
Aluminum Hydroxide Gel .....400 mg.
Magnesium Oxide .....200 mg.
Sodium Lauryl Sulfate...25 mg.
Methylcellulose .....100 mg.

Dosage:

Gel — 2 to 4 teaspoonfuls every three hours, or as needed. Tablets — 2 tablets (chewed for more rapid action) every three hours, or as needed.

Supplied:

Gel — 12 oz. bottles. Tablets — bottles of 100 and 1,000.

1. Johnston, R.L.: J. Indiana St. M.A. 46:869, 1953. 2. McHardy, G., and Browne, D.: Southern M. J. 45:1139, 1952.

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Dosage: May range from 6.25 to 50 mg. daily, taken in 1 or 2 doses. Many patients experience a diurnal variation in mood—marked by morning depression. Under such circumstances, a single dose in the morning is often sufficient.

Supplied: Tablets, 25 mg. (scored), vials of 24 and bottles of 100; also, 12.5 mg. (scored), bottles of 100. Elixir, 25 mg. per 5-cc. teaspoonful, bottles of 1 pint.

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increasing the peripheral resistance without increasing output, the latter becomes a definitely superior drug in the treatment of the type of shock under discussion.

• CARCINOLYTIC DRUGS

Q. What is the present view on carcinolytic drugs like H 11 extract, Laetrile and chinone with ethylene-imin? Is it advisable to combine them with CORTONE®? Is there any institution that has tried these carcinolytic drugs?

M.D.—Philippines

A. H 11 has been carefully evaluated by a commission of the Medical Research Council of Great Britain and was found to be of no value. The claims made for it by its originators could not be accepted, even with regard to animal studies, since a tumor was used which regressed spontaneously (without H 11 treatment) in 36 of 58 mice. The ethylene-imins, on the other hand, are of real value in the palliation of selected cases in the lymphoma group. It has recently been suggested that combination of these latter compounds with cortisone in therapy yields slightly superior results.

• TONSILLECTOMY IN YOUNG CHILD

Q. A two year old child has had repeated attacks of tonsillitis during the past year. Various antibiotics have been administered to lower her temperature and to treat the infection.

Should the tonsils be removed at this time, or, if not, how long should we delay in doing so? I am fully aware of the danger which may be involved in administering an anesthetic to a two year old child, but have never understood why it is considered much safer to wait until the child is two and a half or three years of age.

I shall appreciate your comments and advice.

M.D.—Texas

A. Indications for tonsillectomy and the age at which tonsillectomy should or should not be carried out have been the subject of considerable controversy. There is reasonable agreement that the indications for tonsillectomy are: (1) repeated attacks of tonsillitis, (2) repeated attacks of cervical adenitis, (3) interference with respiration, deglutition or phonation because of the size of the tonsils, (4) uncommonly, when the need for removal of foci of infection is present and the tonsils are found to contain pus, and (5) rarely, when tonsils are thought to be a source

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of infection in patients who have tuberculous cervical adenitis.

If these are accepted as indications for tonsillectomy, the age of the patient should not be a reason for withholding surgical treatment.

The risk of anesthesia in young children can be greatly decreased if the proper practices and procedures are followed. This subject has been well presented in an article by Hallberg and Pender entitled "Endotracheal anesthesia for tonsillectomy and adenoidectomy in children" (J.A.M.A. 153:1073-1074 [November 21] 1953).

• FINGERNAIL DISCOLORATION

Q. A 38 year old hairdresser, otherwise in good health, noted the onset of discoloration of her fingernails about six months ago. Since then this brownish tinge has become more marked and now involves all of her nails. In addition, the nails have lost their normal luster, are thin and brittle and now are beginning to curl up at the ends.

I realize there are many possibilities, e.g., fungous infection, chronic irritation from hair-dressing solutions, etc., but she volunteers the interesting observation that there seems to be a definite relationship between the progress of nail changes and an increase in her use, or her customers' use, of a selenium sulfide preparation for the hair and scalp.

Could you (1) offer a diagnosis from this outline; (2) tell me if chronic selenium poisoning could cause such localized changes only; and (3) suggest therapy (she is now on 10 per cent ammoniated mercury ointment daily and constant use of surgical rubber gloves)?

M.D.—British Columbia

A. The description of the nails having lost their luster, becoming brittle and having a brownish tinge makes one think of nail polish sensitivity, since most beauty parlor operators have their hands in water a great deal. This makes me suspect that she uses some nail base or coat to keep the polish on. Sensitivity to plastics in these bases sometimes is very severe, causing loss of the entire nail.

I have never heard of selenium poisoning causing any localized changes.

I would suggest the withdrawal of all nail polish and a check on the basal metabolism rate. If it is low, thyroid, 1½ gr. daily, is indicated.

ELEMENT OF BIOLOGICAL NECESSITY,
upon which man's dependence is revealed by continuing research in metabolism.

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Inadequate iodine, coupled with mild hypothyroidism, is characteristic of the age group over forty, whose most conspicuous complaints are chronic fatigue, poor memory and sleeplessness. Evidence indicates even a mild iodine poverty plus hypothyroidism may produce cumulative harm, contributing to hypercholesterolemia, myocardial damage and mental regression.

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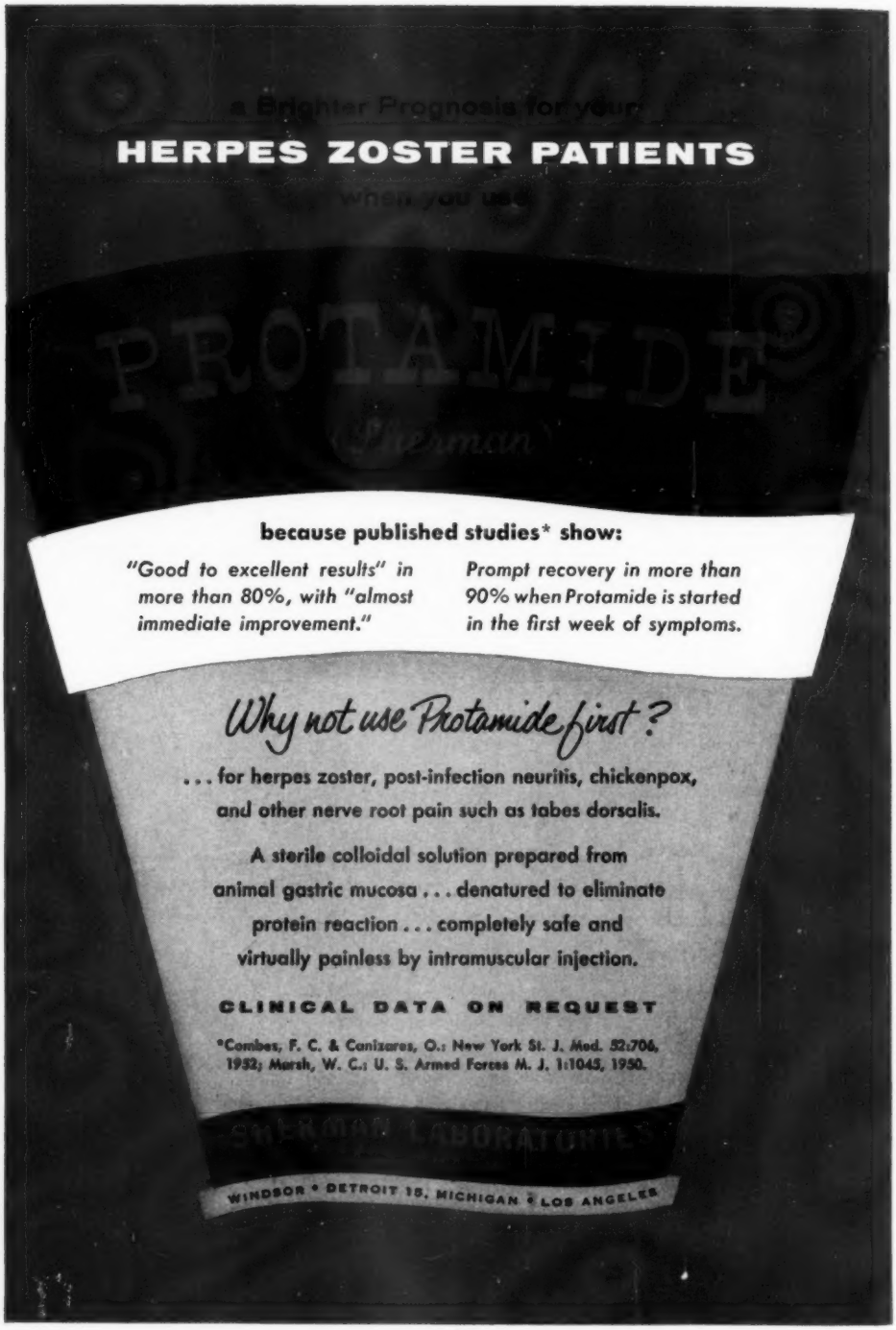
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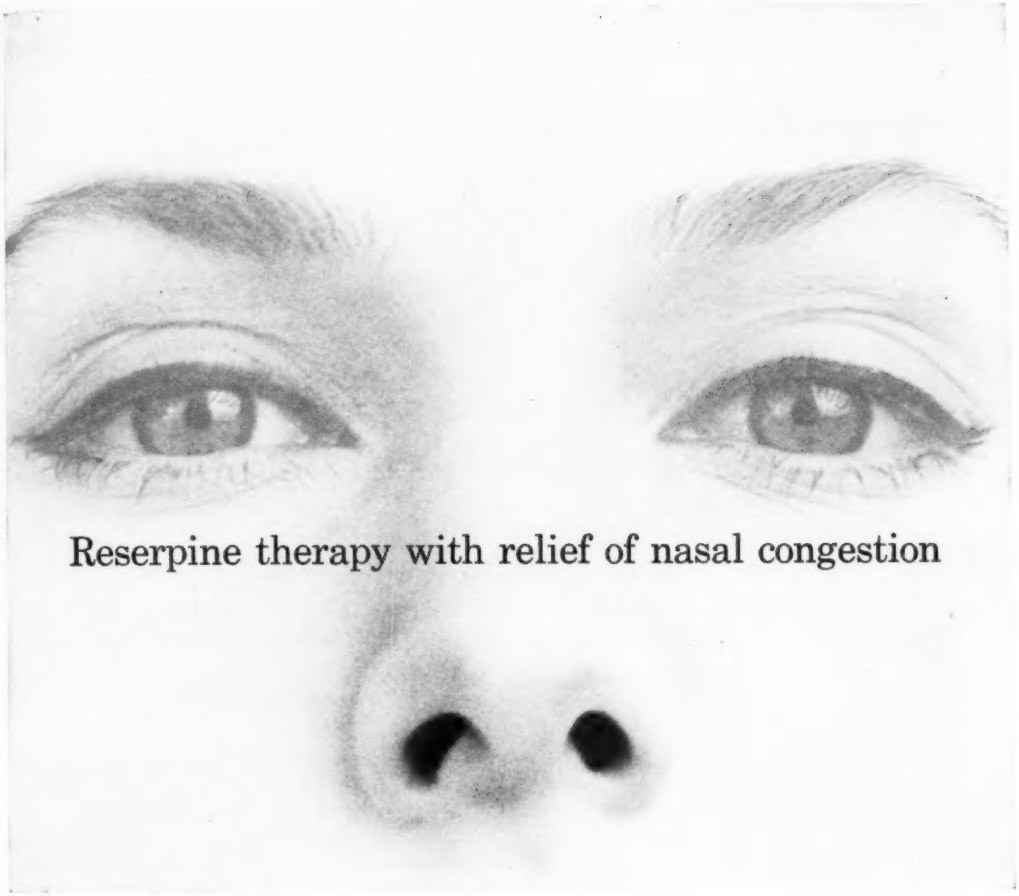

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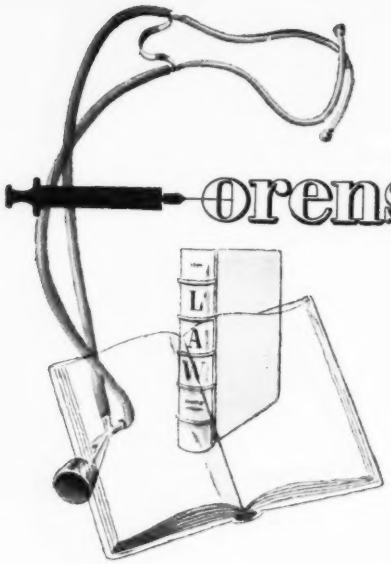

Forensic Medicine

Editors

Dr. Thomas A. Gonzales
Dr. Milton Helpern
Dr. Samuel A. Levinson

Psychiatric Evidence in Legal Tests of Insanity

BURTRUM C. SCHIELE* AND MONRAD G. PAULSEN**

University of Minnesota, Minneapolis

The Legal Rules

Acts just and unjust being as we have described them, a man acts unjustly or justly whenever he does such acts voluntarily; when involuntarily, he acts neither unjustly nor justly except in an incidental way: for he does things which happen to be just or unjust."

These are the words of Aristotle written centuries before Christ. That praise or blame depends upon the will and intention of him who acts is an attitude deeply ingrained in the Western if not the whole world community. "I didn't mean it" is the first defense of child and adult.

For a long period the most serious crimes in the common law have consisted not merely of a harmful act but also of a certain subjective state. A criminal intention is required bearing the mysterious Latin label *mens rea*. Fundamentally the defense of insanity in a criminal case is an argument that the accused was incapable of entertaining the intention demanded by law.

A little over a century ago the English judges made an attempt to describe the kind of mental disorder which would satisfy the courts that an accused could not formulate a criminal design. The judges in M'Naghten's case said:

". . . to establish a defense on the ground of insanity, it must be clearly proved that at the time of the committing of the act, the party accused was laboring under such a defect of reason, from a disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong."

We should note that the disorder described is said to be a defect of reason. The emphasis is on a limitation of cognitive power. It is a rule that we inherit from a psychology which distinguished sharply between the "faculties" of the mind. Under the M'Naghten rule, testimony about many kinds of serious mental disease would be logically irrelevant because in some cases the disease may well have left the capacity to know relatively unimpaired. However, it should be added that in practice the physician is sometimes given greater latitude of testimony than the rule would suggest. The logic of such liberal interpretation is grounded in understanding that the soul of man is one, and impairment in one respect is

(Continued on page A-36)

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usually associated with handicap in another.

The M'Naghten rule, in common with all other legal tests of insanity, points to mental condition at the time, indeed at the moment, of the act. It is the sanity of the accused at that precise instant of time which is the issue in a criminal case. Lack of mental capacity plays a role in another part of the criminal case also: A person will not be tried if he is insane. Insanity for this purpose has its own special test: Is the accused competent to understand the nature of the charges against him and to participate rationally in his own defense? Mental incompetency at the time of the trial differs sharply in its consequences from incompetency at the time of alleged crime. A person who is insane at the time of the trial may be tried when reason has been restored. A person who is insane at the time of the act is not guilty. There is one more criminal law rule about insanity which seems barbarous. An insane person convicted of a capital offense which he committed while in his right mind will not be executed until the return of reason.

In some states the right-wrong test of M'Naghten has been supplemented by the doctrine that if the allegedly criminal act was the result of an "irresistible impulse" the accused should be acquitted. This test of mental disorder refers to defect of will power just as the M'Naghten rule refers to the impaired power of knowing. The test is difficult to apply even when we attempt to do so sympathetically. The books are full of cases telling of impulses which were not resisted but were they really irresistible? Would the accused have yielded to his weakness if a policeman had stood by his side? Further, as one judge has observed recently, the irresistible impulse test implies that mental disease produces "only sudden, momentary or spontaneous inclinations to com-

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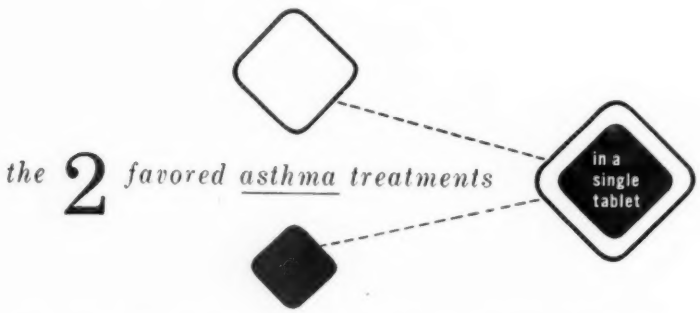

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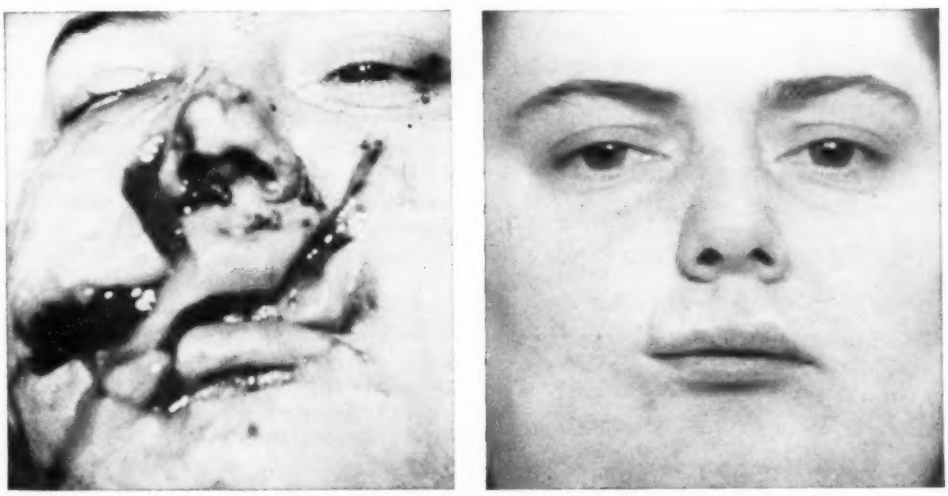
*Straith, C. L., and Straith, R. E.: Detroit, Michigan. Postgrad. Med. *14*:165, Sept., 1953.



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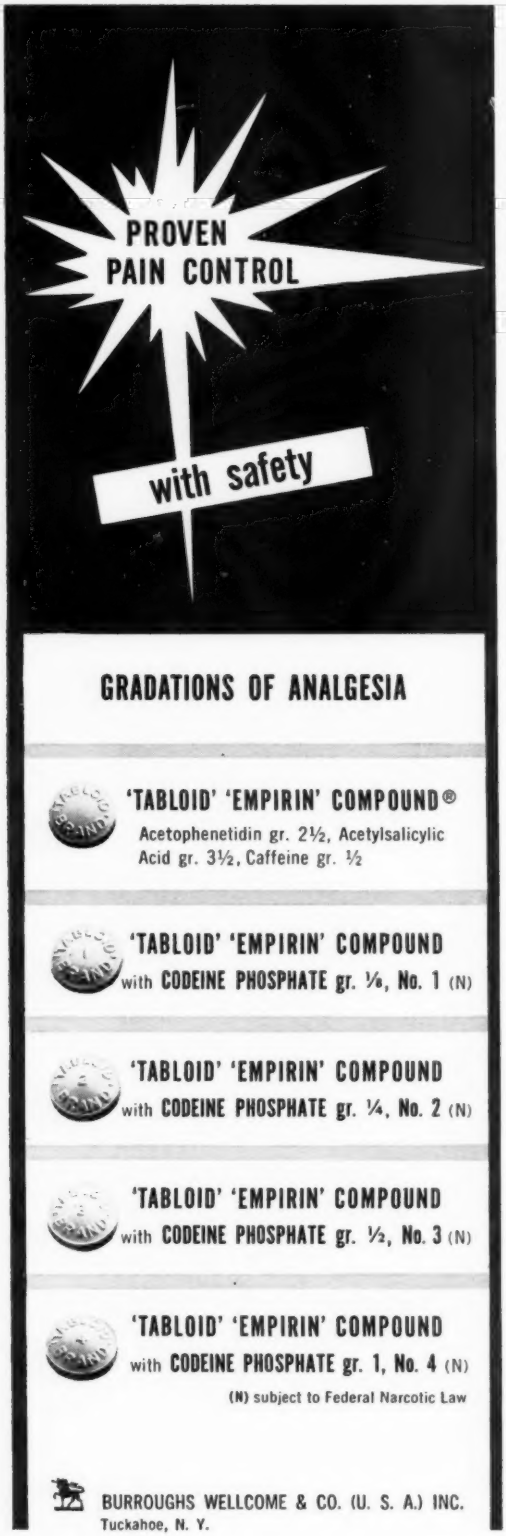
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mit unlawful acts" and "gives no recognition to mental illness characterized by brooding and reflection."

In 1954 the Court of Appeals for the District of Columbia found both tests inadequate.[1] A broader test for acquittal was formulated: An accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect. The answer to the question put by what we may call the "product-of-disease" test is given by a jury.

This approach gives much more scope for the testimony of the psychologist and psychiatrist. They can describe the patient's illness in medical terms. The question is simply: Was the disease present and was the criminal act a product of that disease? The witness will be permitted to tell of the accused's cognitive powers, his ability to control his impulses, the presence of delusions, psychologic test scores, indeed, to testify about any symptom of mental disease. The test still requires us to recognize disease and distinguish that state from health. The jury is still put to the task of relieving of responsibility those possessing a diseased mental condition and of holding accountable those who possess mental health. The jury must make difficult decisions on the troublesome issue of causation. Some will fear that the product-of-disease test takes us far toward a terminal position which would link all crime with an unusual personality characteristic—a characteristic operating to excuse the person from punishment.

The stated rules may be much more doubtful than the results actually reached under them. Insanity is a defense common only in serious cases which are ordinarily tried to a jury. Control of the jury in a criminal case is accomplished largely through giving of instructions by the judge. These instructions tell the jury of the test for insanity which the jury then applies to the evidence. In reaching their verdict undoubtedly jurymen consider matters other than the technical issues as those issues are presented in the instructions. Inevitably the jurors give a kind of common sense judgment about the mental state of the accused—a judgment which, when guided by expert testimony, may be as sound as earthbound creatures can reach. Perhaps better for us than the wisdom of its judgments is the jury's responsibility for them—a responsibility which is diffused when the jury disperses. No identifiable individual need shoulder public blame for errors committed.

The Medicolegal Problems

Psychiatrists and lawyers alike are unhappy with the M'Naghten rule. Henry Weihofen[2,3] introduced his book, "Insanity as a Defense in Criminal Law," with the statement:

"Probably no branch of the criminal law has been the subject of so much criticism and controversy as the defense of insanity. It is charged that the rules of law governing insanity as a defense to crime are vague and confused; that in so far as these rules are clear, they are clearly unsound, in that they are based upon notions of mental disorder discredited by modern science; and that the procedural machinery for trying cases where this defense is raised is inefficient and blundering in its results."

The M'Naghten rule is not a test of mental disorder. Almost everyone can pass the right-wrong test in a sense. The only exception would be grossly deteriorated and delusional psychotics or persons with severely defective intellect, but these seldom have a chance to commit crime. Probably all murderers know what they are doing and understand the consequences of their acts. Thus they are legally sane even though they may be suffering from a clear-cut mental disorder. The nature of mental disorder is such that the disturbed individual may know what he is doing but, being impelled by unconscious emotional forces, may not know why.

In commenting on this situation, the Forensic Committee of the Group for the Advancement of Psychiatry (GAP)[4] reports:

"This consideration poses a difficult problem for the psychiatrist as witness under the present system wherein he must answer questions according to a tacitly understood convention. When the 'knowledge of right and wrong' or 'of the nature and quality of the act' question is put, the psychiatrist witness knows that if he answers 'yes' he is saying that the defendant is legally sane whereas, for example, he may know from his technical insight and careful examination that the defendant is mentally ill and has committed his offense out of unconscious need of punishment. This leaves the witness with his intellectual honesty at stake and with no escape. For suppose he answers 'no'—the defendant did not know the difference between 'right and wrong.' Now he has given the 'legal' answer which conveys the psychiatric truth: the defendant is mentally ill. Next comes the cross-examination and the psychiatrist finds that he cannot relate any vital in-

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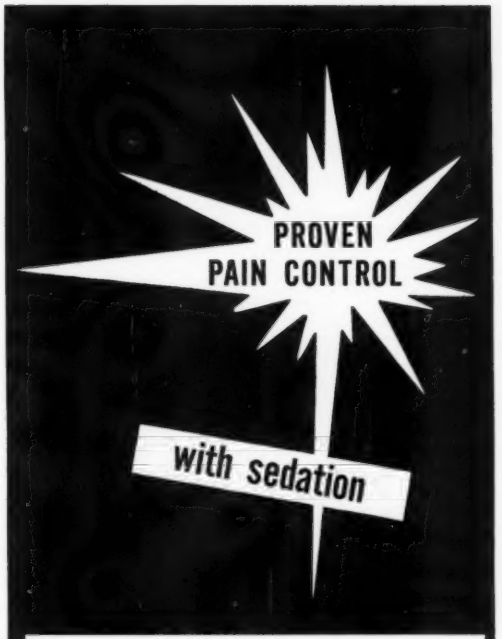

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formation about the defendant without contradicting himself. Such an imposition is not expressly intended by the law in words; it is nevertheless inherent in the law in action. . . . Only in cases of disturbed consciousness or of idiocy can the psychiatrist make honest replies to the M'Naghten test questions."

Just as the rules do not make it easy to properly evaluate and institute treatment for the mentally disturbed, they do not assist the law in safeguarding society. Regarding this point, the GAP Committee[4] went on to say:

"Many dangerous persons are essentially undeterrable and it is the business of psychiatry to supply the law with such knowledge as is necessary to detect such persons among whom may be counted several classes of mentally disordered offenders. In the trial, the M'Naghten formula does not determine realistically who such persons are. Many undeterrable offenders are treated as if they are 'sane' and sent to prison to be released at the termination of sentence, free to repeat the cycle. . . . as a device of criminal law administration, the Rules touch only a fraction of undeterrable mentally ill. In this there is no security for the law-abiding community."

In this connection it may be well to mention that, contrary to what some persons seem to think, psychiatrists do not view most criminals, let alone all of them, as being mentally ill.

Recent developments concerning the product-of-disease test have aroused a great deal of interest among psychiatrists. If generally adopted, this test might allow a fuller and more fair application of modern psychiatry to the problems of criminal law. The advantages of the product-of-disease test over the right-wrong test may be illustrated by the following case:

We were asked to examine a young mother who had murdered her own two month old infant. The patient was able to describe clearly her behavior and her feelings before, during and after the tragedy. She related that she had always wanted three children and that this baby was planned. However, three months before its birth she began to have one of her nervous periods. After the birth she felt tired and "unreal" and began to think of the baby as a burden. One day she placed the infant face down in water and allowed it to drown. Except for this criminal act, no one had noted anything unusual or abnormal about the patient, and it was during our examination that she related the story of her nervous

(Continued on page A-42)

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tension and her unreal feelings, *for the first time.* She further related that she had had a number of similar "spells" lasting two to three months each and dating back to childhood. The most severe of these had occurred following the delivery of her second child several years earlier. On this occasion she did tell her family doctor that she was feeling "nervous" and "nerve pills" were prescribed. She improved in a few weeks.

We studied the patient intensively over a two month period and arrived at the conclusion that she had shown no evidence of psychosis during our examination or in the past. Using the traditional right-wrong test, the patient knew that she was killing her baby by placing it in the water. In our opinion, at the time of the act she knew that what she was doing was wrong. Thus we reported to the judge that in our opinion, by applying the right-wrong test, she was not insane at the time of the crime. On the other hand, it would have made us very uncomfortable if we would have had to restrict our opinion to the right-wrong test. In order to give the court the full benefit of modern psychiatric thinking we reported in part as follows: "We find the patient to be suffering from a schizoid personality disorder. In other words though responsible under the application of the right-wrong test, she is not normal. *Her criminal act was committed as a product of her psychiatric illness.*

"In this instance, the patient did not develop an overt psychosis nor did she resort to suicide or other self-punitive measures. She did not conceive of seeking help nor of letting her distress be known. She reacted by destroying her baby. This attempt at a solution of her problems was a symptom of her psychiatric illness."

Thus, using the product-of-disease test as an example we reported our full opinion to the court and managed to convey the point that, even though the patient was able to pass the right-wrong test, she was still mentally disordered. The judge referred the case to the grand jury, a "No-bill" was returned; the patient was then committed to a mental hospital by the probate court.

Were it not for the approach possible in the product-of-disease test, any psychiatrist would be tempted to testify that this patient was psychotic and did not pass the right-wrong test. In so doing, the psychiatrist would probably be using the occurrence of the abnormal act as the chief evidence of mental disorder and allowing his con-

(Continued on page A-44)

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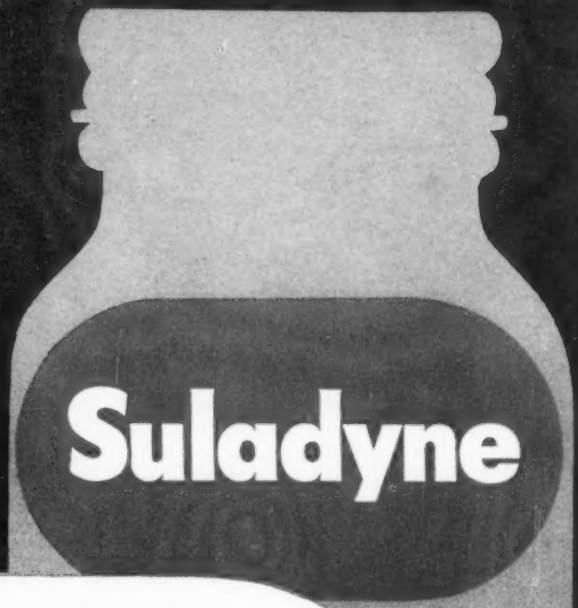

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cern over the disposition of the case to influence his testimony. This is a natural but unfortunate temptation.

Of course, most psychiatrists would agree that prime responsibility for the disposition of accused persons in the criminal process does not lie with them. But a psychiatrist will feel a responsibility if the punishment is visited upon a person because the truth about him cannot be told in full or because the law asks a question to which the physician cannot frame an honest answer. On the other hand, if the specialist can testify in full and in scientifically acceptable terms, he can perform his function without the temptation to distort the truth about one aspect of a patient's condition in order that the patient's entire difficulties may be taken into account.

No matter what legal test is adopted, many troubles face the psychiatrist in court and his testimony has received a great deal of criticism.

The law oversimplifies a lot and operates by absolute categories. It has no "halfway-house." It is not possible for the psychiatrist to divide people into clear-cut categories such as "sane" and "insane" or "mentally healthy" and "mentally diseased," just as it is not a simple matter to divide all people into groups such as the sick and the well or the good and the bad.

To this problem is added the adversary method of the courtroom and the so-called "battle of experts," both so unsatisfactory to the presentation of scientific findings. The sorry spectacle which occasionally occurs is confusing to the public and discrediting to the medical profession. While there is room for legitimate differences of opinion, the battle of experts is largely the result of the legal process. It is really a struggle between lawyers with the psychiatrists being used as weapons.[5] Each side is out to win, and the defendant may be all but forgotten in the process. This adversary procedure is carried out in such a way that each side calls only witnesses favorable to its cause. The witnesses are further questioned in such a way as to bring out the differences between them rather than to search for objective truth.

Some of the difficulties may be illustrated by briefly citing another case:

A youth in his late teens shot and killed his foster mother. While under our study he described in detail the stressful home situation, his increasing hatred for his foster mother, the details of the murder, and his subsequent flight to

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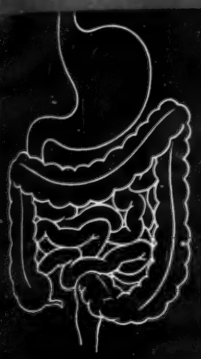

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References: 1. J. Pharm. & Exp. Ther., 98:14, 1950.
2. Gastroenterology, 24:204, 1953.

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another part of the country. He was highly intelligent, socially sophisticated, personable and mature-appearing. In our opinion, he clearly passed the right-wrong test. When his case came to trial, he was defended on the grounds of insanity. He was found guilty of first degree murder and sentenced to life imprisonment. In this process seven experts—six physicians and one clinical psychologist—testified in relation to his mental state: All were called as partisan witnesses and the proceedings were taken over mostly by arguments concerning the right-wrong test. This resulted in the appearance that four of the experts held that he knew the nature and consequences of his act while the other three had exactly the opposite opinion!

In this partisan proceeding we had little chance to bring out subtle but definite abnormalities which we found in this boy. In spite of his outward appearance of maturity and composure, he had shown signs of personal unrest for some time: He was a marked under-achiever in school and in work, he had numerous unfounded medical complaints, and he was preoccupied with thoughts of suicide and with his family difficulties.

Had the experts been allowed to confer with each other and if the full psychiatric picture could have been brought out, as can be done at a medical staff meeting, differences of opinion would not have loomed so large and they certainly would not have attained the degree of polarity which is achieved by the court process.

This, then, is the twin task that psychiatrists would put upon lawyers and lawmakers: First, the legal issues should be defined in such a way as to permit the full presentation and consideration of all the relevant facts concerning an accused's mental disorder. Second, and certainly the more difficult, new ways of presenting this information ought to be found. Some form of communication to the courts should be capable of invention which would permit questions to be explored in a scientific manner and would permit difference of opinion to develop as intellectual disagreements between men of science rather than as battlefield struggles between men of war.

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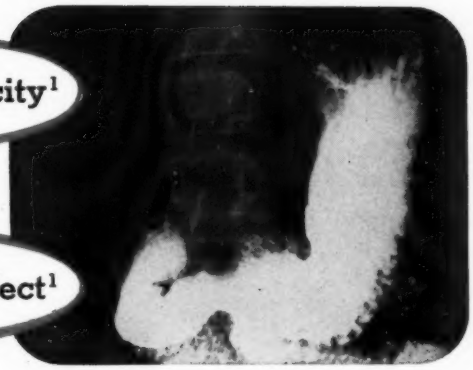

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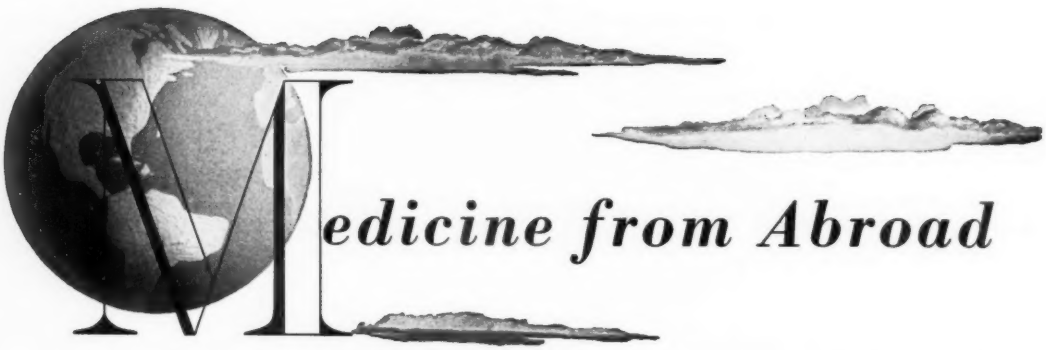
1. Rossett, N.E., Rice, M.L. Jr.: An In Vitro Evaluation of the More Frequently Used Antacids, Gastroenterology 26:490 (1954).
2. Morrison, Samuel: Magnesium Aluminum Hydroxide Gel in the Antacid Therapy of Peptic Ulcer, Am. J. Gastroenterology 22:309 (1954).



Medicine from Abroad

FROM POSTGRADUATE MEDICINE'S CORRESPONDENTS

WEST GERMANY

A visitor's impressions—Rarely does one have the opportunity to visit and observe every facet of life in a country which within a generation has survived two world wars. This opportunity came to me some months ago, when a small group of physicians, surgeons and public health officials were guests of the Federal Republic of Germany and Berlin (West) for a month's study tour of the country.

To an observer from the United States, a nation affected so slightly on its own soil by the ravages of war as compared with European and Asiatic countries, the material and physical destruction experienced by Germany in World War II is almost beyond comprehension.

Despite the crushing losses borne by the Germans of the past generation, the indomitable optimism and never-failing industry peculiar to this people, with material assistance from the United States and the other West German occupation forces, England and France, have produced once again a thriving, alert nation.

When Germany entered World War I in 1914, she was among the leading powers of the world. Her commercial fleet was one of the world's finest. Her educational and cultural advantages were sought by peoples of all nations. She had great scientific research laboratories and led in the manufacture of synthetic products and precision instruments. With Germany's defeat at the end of the first world conflict, her entire economy was thrown out of balance, her manpower depleted, and her educational system disrupted to such an extent that the civilized world suffered.

This observer found Berlin a depressing sight! Where in 1913, the year of our first visit to this German capital, the royal palace, government buildings, library, hotels, beautiful statuary of Unter den Linden, and Brandenburg Thor had stood, only the latter remains. The rest, the former architectural glories of Berlin, is leveled.

Before the restoration program for West Germany following World War II could be started, the newly organized country's public health department had to overcome the rat pestilence which usually follows war destruction. Extreme and systematized methods quickly accomplished the task and rehabilitation was then begun.

Postwar planning commissions have been established in all larger West German cities to supervise reconstruction of bomb-damaged areas. Rebuilding is proceeding rapidly and, in many instances, is resulting in improvement. Widening of streets and avenues, extensive landscaping of public parks and properties and reconstruction are contributing to city-wide beautification.

Medical schools—Of the 18 German uni-

(Continued on page A-58)

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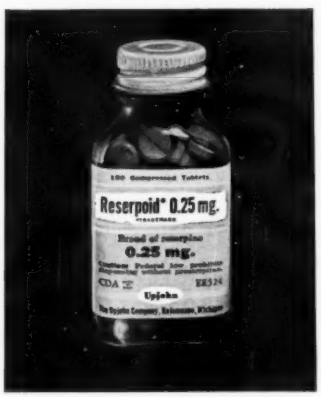


versities teaching medicine, we were privileged to visit nine, where we talked with either the dean or president of each institution. We learned that university faculties had been retarded markedly in the past 25 years due to the disorganization resulting from the first world war, the purge of the Jewish teachers by the Nazis, and, finally, the removal of Nazi party instructors after Hitler's defeat.

We observed that one of the most serious results of war is an imbalance of the population resulting from the heavy mortality of German soldiers. This unnatural annihilation of a large percentage of men puts women in a noticeable majority; hence, illegitimacy is accepted and is so prevalent that to have a child born out of wedlock is no disgrace. The presence of occupation troops helped to increase the population of the country, regardless of color. A singular condition exists in Berlin, where the present death rate exceeds the birth rate.

Vigilance of health departments in various cities has prevented epidemics and spread of disease. City water supplies must be tested periodically; food inspection is rigid; swimming in lakes around the Berlin area is forbidden because of contamination of the water. Garbage and refuse disposal is supervised rigidly.

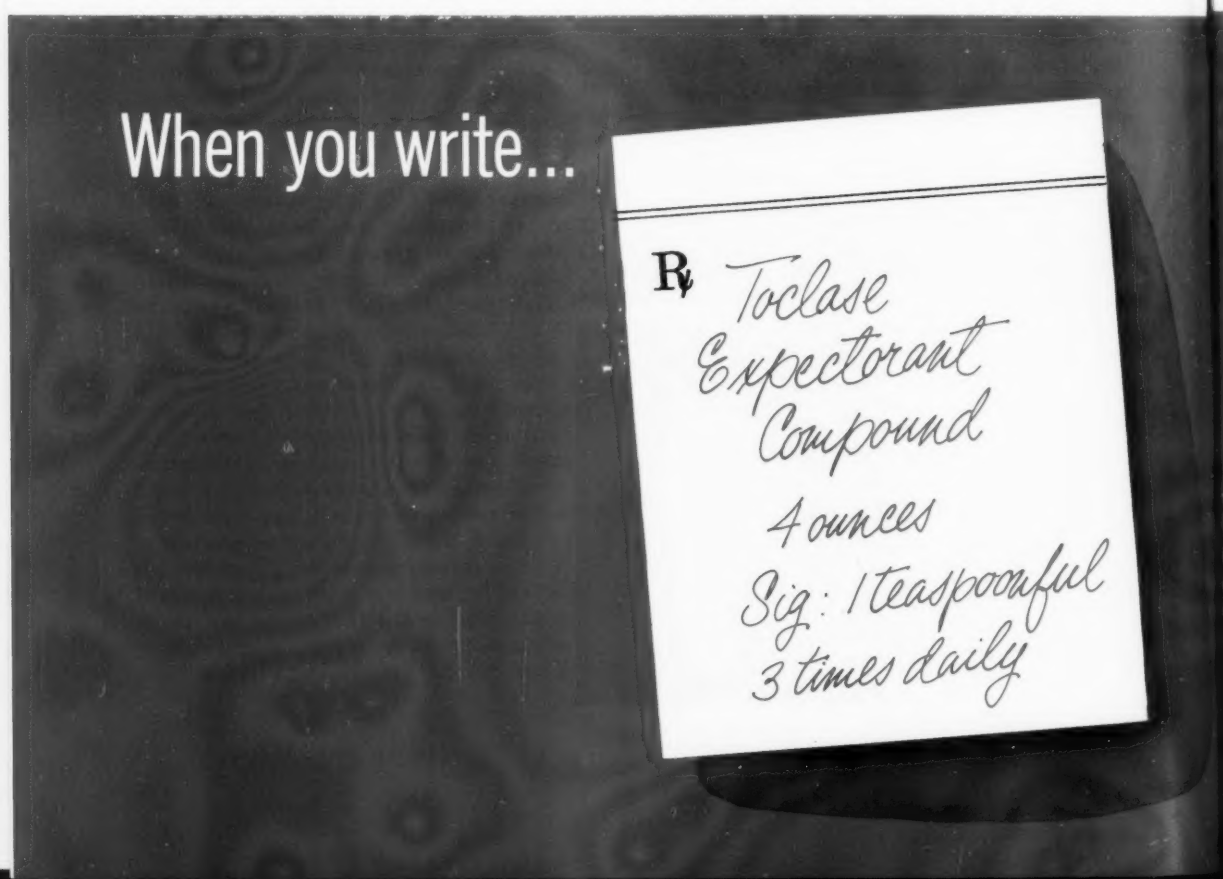
The Federal Republic of Germany was established in 1949 and consists of the British, American and French occupied zones. It covers an area of 245,289 square kilometers. The size of the Federal Republic is thus larger than that of Great Britain and Ireland, but not half as large as that of France.

The population on October 1, 1952 was 48,593,500. It is distributed in nine different *Länders* or states—Hamburg, Bremen, Schleswig-Holstein, Lower Saxony, North Rhine-Wesphalia, Hesse, Rhine Palatinate, Baden, Württemberg and Bavaria.

The president, elected for a five year term, nominates a chancellor who is confirmed by the *Bundestag*. This legislative body of the German people, comprised of 402 members, corresponds to our House of Representatives.

The present chancellor, Konrad Adenauer, 79, leads his country with great strength of character and adroit wisdom. He is greatly beloved and respected.

Spiritual aspects of life have done much to strengthen the German people since the end of the war. At present, the different religious denominations work together more harmoniously



than they have in the last hundred years. Of the West German population, 45 per cent is Roman Catholic and 51 per cent Protestant, with the Lutherans predominating. Each individual Christian is taxed by the government. Atheists, of whom there are few, we were told, are exempt from this unusual levy.

Grand opera, one of the glories of pre-World War I Germany, has been restored. Last February, Giuseppe Verdi's "Nabucco," which Hitler had forbidden, was revived. In Berlin, we had the good fortune to attend a performance of this rarely given work. The opera's stirring chorus depicting a lament of homesick Jews is comparable in beauty to the immortal "Pilgrim's Chorus" from Wagner's "Tannhäuser." So well did the audience receive this chorus of Jewish lament, it was repeated *in toto* by the opera troupe. We also heard a lighter opera, Donizetti's "Don Pasquale," and a new version of "Doktor Faust" by Ferruccio Busoni at the Berlin state opera house.

Symphonic music is again coming into its own. In Hamburg, we heard the city's symphony orchestra with a mixed chorus of 250 voices give the thirteenth century work, "Carmino Romano," first performed by Benedictine monks.

Operetta and legitimate theater productions also are enjoying a revival.

A visit to the city of Giessen and a day spent at the laboratory and home of Dr. Wolfgang Spatz, an eminent neuropathologist, proved to be a high light of the entire study tour. Dr. Spatz had been assigned the mission of reporting grossly and microscopically on the brain of a female body which had been buried 2000 years ago and exhumed in 1953 from a peat bog in Schleswig-Holstein. We held the specimen in our hands and were given the liberty of looking at a stained section under the microscope.

We enjoyed a delectable meal prepared by Dr. Spatz's wife, followed by a recorder concert performed by our hostess and her three daughters. Mrs. Spatz showed us a loom on which in three days she can weave enough cloth for either a son's suit or a daughter's dress. She had woven the cloth and made the dresses her daughters wore that day.

Dr. Spatz, a highly religious and respected man, was held prisoner of war by the Russians during the last conflict. He did postmortems on the soldiers who died in his camp; then before burial of each, said a prayer at the graveside.

(Continued on page A-62)

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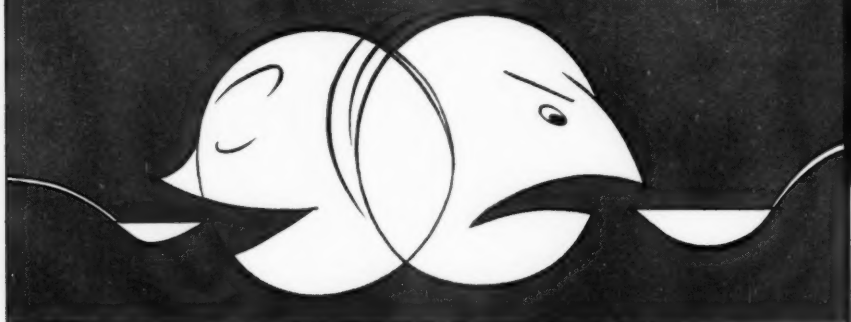

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1. Council on Pharmacy and Chemistry, A. M. A.: New and Nonofficial Remedies 1952, Philadelphia, J. B. Lippincott Company, 1952, p. 311.

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We observed that the majority of the West German populace is well nourished, comfortably dressed, and industriously occupied. The youth, as in America, enjoy vigorous outdoor activities. After a three day snow, the trains were loaded with carefree young people going to the mountains for winter sports. Those interviewed hold no resentment for the actions of their forefathers.

With the type and caliber of men and women who have the future of West Germany in charge, and with the energetic efforts put forth by its citizens, I feel this new nation will soon be a leading world power, one which, if permitted, will join fervently with the other world powers to help establish lasting peace, the only hope of mankind.

Peace means progress and happiness.

War means retardation and suffering.

Aggressiveness for existence and progress is to be commended.

Aggressiveness for power and domination inevitably leads to greed, avarice and destruction.

As expressed previously, it was an unusual privilege to observe a nation which came close to annihilation, rehabilitating itself and apparently enjoying the task.

J. MATHER PFEIFFENBERGER

GENEVA

Scientific contributions of Swiss surgeons —At the turn of the century, Switzerland was a leader in the surgical world, having produced such men as Krönlein, Roux, Girard, Reverdin and Nobel prize winner Kocher. Kocher's fame was world-wide because of the many operative technics which he perfected and which are still used, and especially because of his now-classic research on goiter.

Surgery today is at an almost equal level in most European countries due to the meetings, special publications and personal contacts among the chiefs of service at the universities. What is the contribution and share of Swiss surgery in particular?

The surgical clinic of Geneva University is directed by Professor A. Jentzer, who will retire next year. With his co-workers, Junet, Clerc, Weyeneth and Dubas, he pioneered in various branches of surgery. He was one of the founders of Swiss neurosurgery and performed the first excision of the pituitary in Switzerland. He pio-

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neered in the use of artificial hibernation and splanchnic infiltrations, and had great experience in treating cancer and thyrotoxicosis with radioactive isotopes. Jentzer was first in Switzerland to use manometry and cholangiography during surgery. He recently successfully treated a patient with cancer of the esophagus and pharynx by complete removal of the esophagus and its replacement with the ileum and colon pulled up into the chest. In May 1955 he organized the celebration of the twentieth anniversary of the International College of Surgeons.

The second surgical service of the Cantonal Hospital at Geneva, under the direction of Professor R. Montant, president of the Swiss Society of Surgery, has specialized the last few years in resection of the stomach and in surgery of cancer of the rectum. With the aid of Baumgartner, several members of the staff perfected the technic for the canalization of the great vessels. Calame, one of the pioneers, used laparoscopy in diagnosing hepatobiliary pathways.

The surgical clinic of Lausanne University is headed by Professor P. Decker, successor to the famous Roux. With Francioli and Buffat he published important articles on plastic surgery of the esophagus for treatment of burns, resection of the esophagus in cancer, total gastrectomy, and excision of the lung in pulmonary tuberculosis and lung cancer. His operative technic, always perfect to the smallest detail, aims at reducing surgical risk to the minimum. He recently has successfully operated on aorto-caval aneurysm using his own operative technic. He was also one of the first to reduce the use of antibiotics in intestinal surgery to avoid pseudomembranous enterocolitis, which is caused by *Staphylococcus aureus*, and possible tuberculous complications following gastrectomy.

Outside academic circles at Lausanne, mention also must be made of Paschoud in connection with new and daring abdominal surgery (resection of hepatic lobe, etc.); Naef, first Swiss surgeon to perform mitral valvotomy; and Verdan, who specialized in and published a book on surgery of the hand.

The surgical clinic of Bern University is directed by Professor Lenggenhager, successor to Kocher and de Quervain. His studies have been in the fields of biochemistry and physics. His investigations and experiments enabled him to define the character of the various factors of blood coagulation, and also to discover several new

(Continued on page A-66)

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Milwaukee, Wisc., November 14-17, 1955

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For your convenience, a form is provided at the bottom of this page which should be filled in and addressed to the hotel of your choice. Be sure to send it directly to the hotel.

If the hotel of your choice is unable to accept your reservation, the Milwaukee Housing Bureau will give you the next best accommodations available, providing of course that all available space has not already been taken.

Be definite in the date and hour of arrival as well as the date and approximate time of your departure; also be sure to give the names and addresses of those who are to occupy each room.

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factors, thrombokinine and thrombocatalysine. His studies on high altitudes, oxygen chambers and seasickness have led to discoveries which strongly influenced surgery. He was responsible for the discovery of those types of thrombosis which develop in a liquid medium under rhythmic pressures.

In the field of orthopedics, Dubois and Zollinger have devoted the last few years to treatment of fractures of the femoral neck, brachial neuralgias caused by injuries of cervical vertebrae, muscular stiffness resulting from injury of the knee joint, and the physiopathology of false arthrosis.

Saegesser, of Bern, is the author of a much-consulted handbook entitled "Special Surgical Therapy," the third edition of which is about to be published.

The surgical clinic of the University of Zurich is under the direction of Professor A. Brunner, pupil of the celebrated Sauerbruch. Brunner made Zurich the center of lung surgery in Switzerland. His studies are in all fields of lung surgery, including the different procedures for lung resection and the modern surgery for tuberculosis of the lungs and pleura. He also has made improvements in the treatment of tunnel thorax and diaphragmatic hernia. Others from the surgical clinic whose works are well known are Buff, on plastic surgery; Deucher, on ulcerative colitis, rectal cancer and abdominal surgery; Mayor, on extirpation of the urinary bladder and treatment of cancer of the bladder with radioactive cobalt; and Ricklin, on percutaneous retrograde radiography of the aorta.

Grob, of the surgical service of the Zurich pediatric clinic, was the first to operate on children with congenital malformations of the heart. He also has written articles on treatment of atresia of the esophagus, Hirschsprung's disease and other abnormalities.

The neurosurgical clinic of Zurich University is headed by Professor Krayenbuhl, who has had remarkable technical results in the field of tumors and brain abscesses.

Fehr, bone surgeon at Winterthur, called the attention of physicians to the posterior dislocation of the humerus, a type of dislocation which is often unrecognized.

The surgical clinic of Basel University is directed by Professor R. Nissen, pupil of Sauerbruch, who was appointed after serving as director of surgical clinics in Berlin, Ankara and the

(Continued on page A-70)

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Harris, R.: Ann. New York Acad. Sc. 59:95 (April 30) 1954.

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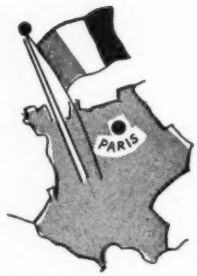
United States. He now is president of the International College of Surgeons. He was successful in performing the first lobectomy in a 12 year old girl with bronchiectasis. His writings on surgery of the esophagus, stomach and lungs are considered outstanding in the field. Other important writings by clinic members are those by Hess, on surgery of the pancreas; Rothe, on conservation of bone grafts; Hugin, on modern anesthesia; and Allgower, on formation of granulation tissue.

The second surgical service, under Professor Heusser, is particularly devoted to the prevention of postoperative disease, to water and salt metabolism, to the problem of proteins, and to acute intestinal occlusion. Heusser was the first to develop a method of peritoneal dialysis which could be used in man, and he is now working on the problem of the artificial kidney.

Since it would be impossible to quote all Swiss surgeons specializing in the various fields, the reader is referred to the great undertaking of Swiss surgeons entitled "Treatise of Surgery," a two volume work published in German and French under the editorship of Brunner, Henschen, Heusser, Jentzer and the memorable Schurch and Veyrassat.

PARIS



Acute and subacute terminal ileitis—Cherigié, radiologist in the Paris hospitals, and co-workers have discovered that many children are operated on for acute appendicitis at the onset of or during an infectious disease, especially measles, when what they have is not acute appendicitis but acute inflammation of the terminal ileum and the mesenteric lymph nodes.

The pathologic changes which occur are hypertrophy of Peyer's patches and infection of mesenteric lymph nodes along the last ileal loop. Roentgenographically this hypertrophy appears in the form of defects, or lacunae, corresponding to the elevations of lymph follicles on the folds of mucous membrane.

Establishing diagnosis by roentgenography is difficult. In one-fifth of 1300 roentgenograms taken in 70 cases, there was no clear definition. In such instances the physician must resort to barium enema, and then forcibly manipulate the

(Continued on page A-74)

in rheumatoid arthritis

now available...the second
new Schering corticosteroid

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ileocecal valve and differentiate the ileal shadow from that of the colon. The intolerance of the last ileal loop sometimes adds to the difficulty.

In younger children the disease produces lacunae only. In older children the loop is wider, and the images of Ross-Golden, those of nonsclerosing ileitis, are observed. In adults, hypertrophic and often ulcerated Peyer's patches are visible.

The authors studied 70 cases in children. Nine of the children supposedly were healthy but three of them showed roentgenographic evidence of inflammation of the lymph nodes. Of the 47 sick children, 37 had lymphadenitis. Thirty-two of the children with lymphadenitis had measles, and 23 of these patients also had inflammation of the lymph nodes. The rest of the 70 children were not suitable for study. In measles the frequency of lymphadenitis in the mesenteric nodes explains the frequency of acute pseudoappendicitis, which in three cases required operation two days after the efflorescence of skin rash.

It is dangerous to deny the indications for surgery and to delay operation on the basis of roentgenographic examination alone. This should be not done unless the patient is kept under continuous observation. Lymphadenitis of the mesenteric nodes is also seen in scarlet fever, various types of angina, pneumonia and in intestinal parasitic disease.

In adults, acute terminal ileitis occurs in typhoid diseases and in tuberculosis of the small intestines which begins with hyperplasia of Peyer's patches. Crohn's disease is difficult to diagnose because at its onset it shows the same signs as acute terminal ileitis (i.e., widening of the loop and lymphoid enlargement). Certain patients who recovered spontaneously probably had simple inflammation of the lymph nodes.

The nature of the lymphadenitis which characterizes terminal ileitis has been under discussion. Several authors consider it the simple result of different inflammatory processes. Others talk of a primary terminal ileitis with secondary involvement of the lymph nodes, and still others consider it a form of tuberculosis. Arnulf and Buffard described primary acute lymphadenitis as an independent disease which is probably due to a virus and which breaks out in the course of various infectious diseases. Reilly found acute lymphadenitis in experimental animals in the course of acute infections. It is sometimes associated with invagination. These changes in animals resemble those observed in man.

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Psychomotor Seizures or Temporal Lobe Epilepsy

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University of Minnesota Medical School, Minneapolis

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INTEREST in the function of the temporal lobe has increased markedly in recent years. Much of this interest has been due to the recognition of this site as the source of that form of paroxysmal activity known as the psychomotor seizure, or perhaps better known to some by the older term of psychic equivalents. Little meaning is conveyed by such terms and, therefore, since this region is the root of this seizure pattern, a more acceptable and appropriate term would be temporal lobe epilepsy. While the various clinical phenomena characteristic of this type of seizure have been known for some time and designated by a variety of names, the advent of electro-encephalography has crystallized these phenomena into the area known as the temporal lobe.

With a knowledge of the intricate connections of this area, one can readily appreciate the diversified pattern of the seizure which stems from this region. The elaborate connections of the temporal lobe bring it into contact with every other part of the brain. This lobe is a combination of the phylogenetically old brain and the new. The former, or primitive brain, is intimately connected with the autonomic nervous system and hence exerts considerable influence on the affective aspects of emotion. With the development of the cortex, or new brain, higher functions of audition, memory, recall and language have been invested within this area, thus immeasurably increasing its complexity. Deep connections with the hypothalamus, the thalamus and the basal ganglia provide contact with structures involved in visceral control, emotional expression, sleep and locomotion. Thus, there exists within the temporal lobe a link between man's highest and most primitive functions.

Within the confines of the temporal lobe pass visual, auditory and vestibular fibers, the

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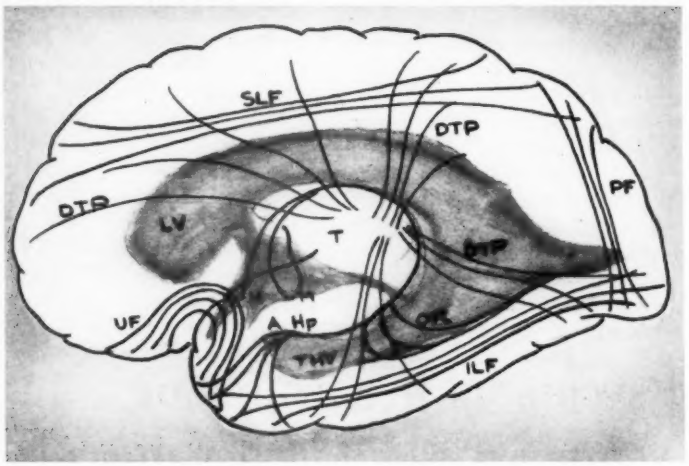

FIGURE 1. Semidiagrammatic view of temporal lobe and its connections. The ventricular system is the shaded area. The complexity of the temporal connections are to be noted.

LV	lateral ventricle	Hp	hippocampus
THV	temporal horn of lateral ventricle	OR	optic radiations
T	thalamus	ILF	inferior long. fasciculus
H	hypothalamus	SLF	superior long. fasciculus
M	mammillary body	PF	perpendicular fasciculus
A	amygdaloid nucleus	UF	uncinate fasciculus

DTP diffuse thalamic projection system

latter two terminating in the temporal lobe cortex. The optic radiations pass through without interruption. More important are the associational fibers which bring the lobe into contact with other areas of the brain. The uncinate fasciculus and the inferior longitudinal fasciculus are two well-developed tracts of this type, connecting the frontal and parietooccipital areas, respectively. There are connections, very complex and devious of route, to the thalamus and the hypothalamus, the latter being the head ganglion of the autonomic nervous system. From the former, projection system fibers radiate diffusely to all parts of the cerebral cortex. A well-developed fiber bundle, the anterior commissure, connects both temporal lobes. Embedded within the medial surface of this lobe are the amygdaloid nucleus and the hippocampus, both of unquestioned though not clearly defined importance, but concerned with emotion and its control. Finally, the ventricular system invades the substance of this lobe in the form of the temporal horn of the lateral ventricle. The propinquity of the sensorimotor systems completes the temporal lobe complex. Figure 1 indicates the chief connections of this vital area. Such a description, however brief, is a necessary prelude to the appreciation of the clinical phenomena arising from this lobe.

Now a word concerning the clinical phenomena. How do we recognize a psychomotor seizure as such? What are its characteristics? It should be stated that a psychomotor seizure, while at best a bizarre, unpredictable combination of motor and psychic activity, is characterized by four general types of phenomena: (1) motor, (2) sensory, (3) autonomic and (4) psychic.

Contraversive and ipsiversive movements, or the movements of the head and eyes to one side; tonic extension of the contralateral and ipsilateral extremities; facial twitching; masticatory, or chewing, movements; purposeless automatic muscular activity of a diverse nature—all constitute the important motor phenomena of these seizures. Visceral phenomena include epigastric and thoracic sensations,

salivation, palpitation, pallor, flushing, defecation and micturition. Sensory phenomena are common in this type of seizure and include olfactory, gustatory, auditory and visual sensations, vertigo and dizziness. The psychic phenomena include expressions of fear, fright, anger, hate, confusion, disorientation, hallucinations of one type or another, assault, and fugue states. Amnesia, usually present, is not a constant feature. Table 1 outlines the clinical features of the temporal lobe seizure.

Preseizure sensations, or aurae, may also be motor, sensory or psychic in nature. These are outlined in table 2 and the relative frequency of the various aurae in a group of 48 cases is indicated. Examination of this table demonstrates the frequency of sensory and autonomic aurae. The latter are the most frequent, comprising 47 per cent of the group. The recognition of abdominal pain and epigastric distress as a cerebral symptom may forestall unnecessary abdominal operations. Fainting, syncope, sudomotor activity and pilo-erection are phenomena experienced as a prelude to an attack.

TABLE 1

SYMPTOMATIC INDICATIONS OF TEMPORAL LOBE SEIZURES

1. SOMATOMOTOR PHENOMENA
   - Adversive movements of head, eyes and body (contraversive and ipsiversive)
   - Tonic extension, contralateral extremities
   - Contralateral facial twitching
   - Masticatory movements
   - Purposeless automatic muscular activity
2. VISCERAL PHENOMENA
   - Epigastric and thoracic sensations
   - Salivation
   - Palpitation
   - Pallor, flushing
   - Defecation, micturition, pilo-erection
3. SENSORY PHENOMENA
   - Olfactory sensations
   - Vertigo, dizziness
   - Gustatory sensations
   - Auditory and visual sensations
4. PSYCHIC PHENOMENA
   - Expressions of fear, fright, anger
   - Assault
   - Disorientation
   - Hallucination, déjà vu, jamais vu
   - Fugue states

TABLE 2

RELATIVE FREQUENCY OF RECOGNIZED AURAE IN TEMPORAL LOBE LESIONS

TYPE OF AURA	CASES	PER CENT
Motor phenomena	6	12
Sensory		
Auditory	0	0
Gustatory	2	4
Olfactory	1	2
Visual	0	0
Vertiginous	7	12
Autonomic		
Abdominal	13	27
Cardiovascular	6	12
Syncopal	2	4
Pilo-erection and sudomotor	2	4
Psychic		
Perceptual illusions	4	8
Fear and terror	2	4
Hallucinations	0	0
Cephalic sensations	3	6
TOTAL	48	

As an example of the psychic aura, perceptual illusions, fear and terror, automatism, hallucinations and assault have been observed. The latter, not uncommon, is dangerous and an indication for thorough organic investigation. Unfortunately with the coexistence of amnesia, such patients are placed in criminal and psychiatric institutions without consideration of this possibility.

The aura assumes more importance in this seizure pattern than in any of the other types of seizure. The patient, often amnesic, can offer little information. At best the relatives are poor witnesses, most being too frightened to have observed the crucial part of the seizure, namely, its onset. Because of the frequency of the aura, particularly of the autonomic type, many of these persons appear in the offices of surgeons and internists with complaints of abdominal pain, or of lesser degrees of epigastric distress, and subsequently are referred for cerebral investigation, particularly when studies of gastrointestinal and cardiovascular systems are unrevealing.

Individuals with temporal lobe abnormality

may have, in addition to psychomotor seizures, petit mal and generalized convulsions. As a rule, if the deep midline structures are selectively activated by a temporal lobe focus, the individual will experience a petit mal or minor seizure. If the entire cerebral cortex is discharged from a temporal lobe focus, the individual develops a generalized or grand mal seizure (major seizure). This is an important fact to grasp. Too often we become careless in our appreciation of the seizure and are prone to treat it with indifference. This is indeed an error and is tantamount to examining the heart without a stethoscope. In reality, a seizure, if properly analyzed, contains a wealth of information which must be obtained by judicious interrogation.

The frequency of the various seizure patterns with temporal lobe foci is shown in table 3. Grand mal seizures are the most frequent, with the psychomotor seizure itself next in frequency, followed by petit mal, syncopal episodes and diencephalic seizures.

In the interseizure period, the incidence of personality disturbances is worthy of note. In one of the largest series to date, 38 per cent of patients with temporal lobe electro-encephalographic abnormalities had associated psychiatric disorders. In our series of 119 cases, which we recently analyzed, 18 patients warranted a diagnosis of a psychiatric disorder including schizophrenia, psychosis, psychopathic states and a variety of neurotic disturbances. It is of interest to note that three of the patients were in prison for major offenses, two of them for murder.

TABLE 3

FREQUENCY OF SEIZURE TYPES ENCOUNTERED IN 112 CASES OF TEMPORAL LOBE EEG ABNORMALITIES

TYPE	CASES	PER CENT
Grand mal	40	35
Psychomotor	38	33
Petit mal	12	10
Syncopal	12	10
Focal	6	5
Diencephalic	4	3
TOTAL	112	

As far as electro-encephalography is concerned, it is sufficient to say that the electro-encephalographic pattern of temporal lobe epilepsy is usually recognized without difficulty. There are spikes with varying degrees of slow wave activity arising from one temporal lobe or the other and often from both sides. Examples of the electro-encephalographic pattern in temporal lobe epilepsy are shown in figures 2 and 3.

The frequency of electro-encephalographic patterns is shown in table 4. Most of the abnormal discharges in this group have ap-

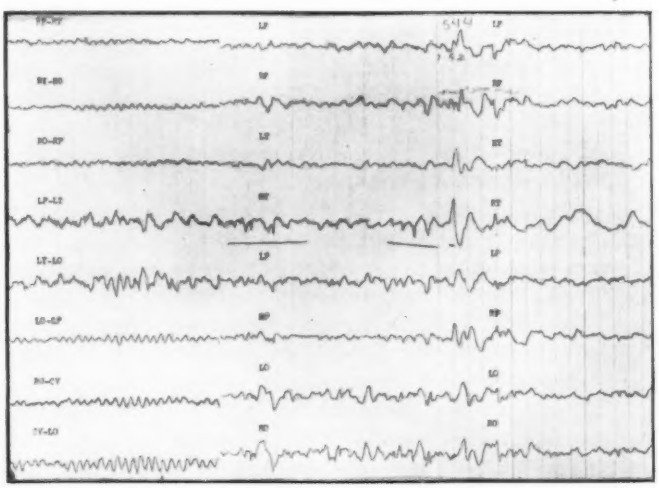

FIGURE 2. Electro-encephalogram in temporal lobe seizure. Channel 4, recorded from the posterior portion of temporal lobe on left, indicates slow wave and spike pattern.

TABLE 4

WAVE TYPE AND LATERALITY IN 119 CASES OF TEMPORAL LOBE DISCHARGE

TYPE	PER CENT
Spikes, left	32
Spikes, right	20
Slow, left	14
Slow, right	4
Spikes, bilateral	14
Slow, bilateral	7
Sharp, left	3
Sharp, right	1
Diencephalic	0.9

peared on the left side with a somewhat smaller percentage arising on the right side. Of special significance is the large percentage of activity appearing bilaterally. Such an event requires the use of special electrographic "activation" procedures, including sleep, METRAZOL®, PENTOTHAL® and photic stimulation in an attempt to lateralize the focus. One should never rely on a single electro-encephalogram in dealing with temporal lobe seizures as this may be quite misleading.

From the standpoint of therapy what has one to offer these patients? There is no seizure type which remains so refractory to treatment as the temporal lobe seizure. The usual anticonvulsant preparations are often unsatisfactory. The majority of these individuals are young people in the second and third decades of life, and their seizures have been inadequately controlled for several years. Many have been unable to find employment because of the seizures. In a recently analyzed group of 119 cases, a 25 per cent reduction in the seizures was obtained in only 17 cases by the use of anticonvulsant drugs. The remainder were benefited to a much less extent. It is apparent then that current anticonvulsant medication, whether used singly or in combination, is ineffectual. One reason for the ineffectiveness of these drugs is to be found in the frequency of neoplasms and degenerative changes that underlie the clinical symptomatology. Table 5 lists the etiologic factors in a group of 48 cases.

TABLE 5

ANALYSIS OF ETIOLOGIES OBTAINED IN 48 CASES OF TEMPORAL LOBE EEG ABNORMALITIES

ETIOLOGY	CASES
Trauma	15
Encephalitis	10
Neoplasms	6
Vascular	5
Congenital defects	3
Degenerations	2
Miscellaneous	7
TOTAL	48

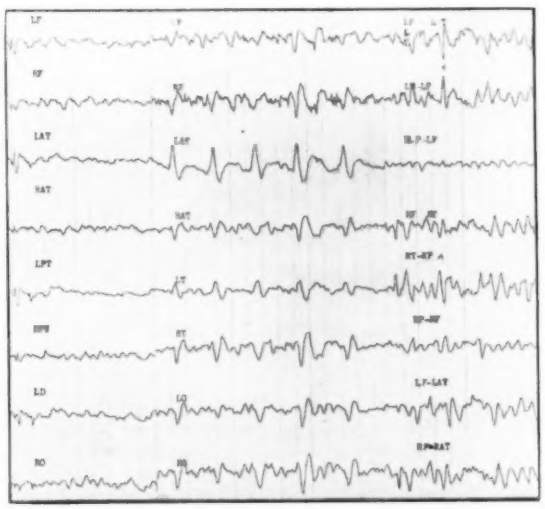

FIGURE 3. Electro-encephalogram in temporal lobe seizure. In channels 3 and 5 recorded from left temporal lobe, upward deflection of high amplitude sharp waves are to be observed, while in all other channels the deflection is downward at the same instant. This is due to the method of electrode arrangement and connections to the amplification system. The wave form and frequency are often observed in temporal lobe seizures.

In recent years, a surgical approach to this problem has been attempted. Impetus to this form of treatment has been given, first of all by the results of surgical therapy; second, by the disappointing results of medicinal therapy; and third, by the surprising incidence of unsuspected tumors and other pathologic processes within the temporal lobe.

Since 1949, Dr. Lyle French, of the Division of Neurosurgery, and I have explored and operated on 20 patients, the majority of them within the past year. Our first patient was operated on in 1950 and has remained free of seizures since that time. No abnormalities were uncovered in our work-up which included pneumoencephalography and angiography. Our surprise at finding a tumor in the first patient stimulated us to operate on additional patients and to investigate this disorder further. In a group of 20 we have found five unsuspected tumors within the temporal lobe for which we had no previous clinical evidence.

Our criteria for surgical treatment have changed considerably. Initially, a period of six months of anticonvulsant medication was advised with the expectant hope of controlling the spells. We no longer regard this as an important part of the program, since all of the patients for whom this was recommended returned without any evidence of control.

Selecting the proper side for surgery presents problems in those instances where clinical data are lacking, and confusion results from bilateral abnormality indicated by the electro-encephalogram. Bilateral electro-encephalographic abnormality appeared in 14 per cent of the cases. It is necessary to resort to activating procedures during electro-encephalography in an attempt to enhance its value. With the additional aids, it is possible to lateralize the process in many cases. In our experience Pentothal sodium has been the most effective activating agent. For some reason, which is the object of our interest, the patient with temporal lobe seizures exhibits an abnormal metabolism of Pentothal. While large doses of Pentothal are required to produce anesthesia, this period is short and the drug is metabolized rapidly. Figure 4 illustrates the necessity of electrographic activation. In this case the waking record was normal, but under the influence of Pentothal high amplitude spikes appeared in the temporal lobe on the left side. The presence of a spike discharge on the opposite side, however, clouded the site of the focus, which was subsequently shown to be on the right side and found by electro-encephalography and delineated further by means of depth electrography.

Cases have arisen in which such technics have failed to lateralize the disturbance. In such instances we have adopted a new procedure of introducing small electrodes into the temporal lobe on both sides. Such electrodes may be left in place for two or three weeks with little harm to the patient, and from the

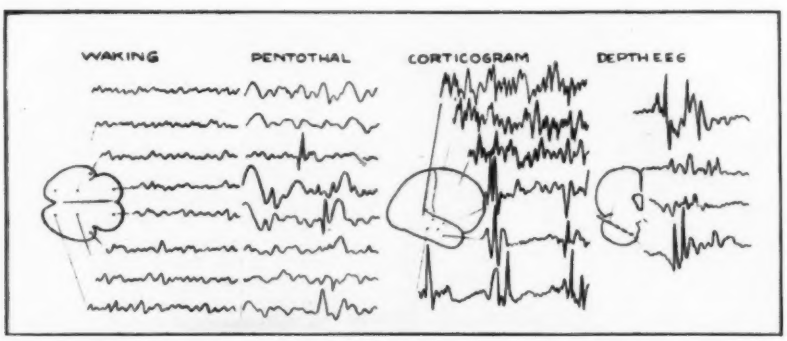

FIGURE 4. Electrographic recording from scalp showing effect of Pentothal in temporal lobe seizures. Electrocorticographic and depth electrographic studies indicated focus in substance of temporal lobe.

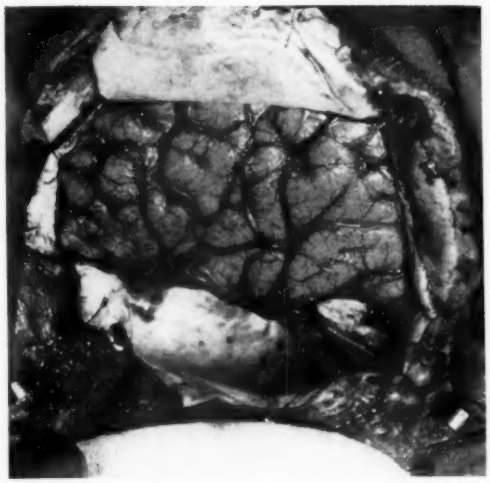

FIGURE 5. Operative field at end of temporal lobectomy demonstrating extent of excision. In lower part of operative site the dural lining of the middle fossa is evident.

electrodes one has the opportunity of recording the cerebral activity over several days in order to obtain a lateralized focus. At the same time it is possible to stimulate the brain and to study the activity and responses to electrical stimulation. In two recent cases such a technic has enabled us to lateralize the disturbance correctly, and in each instance a gross pathologic defect was observed at operation. Of the 20 patients explored, the offending temporal lobe was removed *in toto* in all. Figure 5 illustrates the extent of the excision which includes approximately 9 cm. of the temporal lobe. There has been but one operative death, as a result of a hematoma. Two patients have persisted in having their original seizure pattern; 11 patients have been relieved of the original psychomotor seizures entirely. Two patients have continued to have petit mal seizures, but these have not been troublesome. In the rest, although seizure-free, insufficient time precludes evaluation.

I have touched only lightly on this problem. We are very cognizant of the limitations of such a small series. It is our hope, however, that by focusing your awareness on this problem, the proper diagnosis may be reached earlier and treatment carried out, since we believe that at this time surgery offers the best hope for individuals with this type of paroxysmal activity.

MEETINGS *and Postgraduate Courses*

AMERICAN COLLEGE OF PHYSICIANS POSTGRADUATE COURSES, AUTUMN 1955: Congenital heart disease, November 14-19, at Johns Hopkins Hospital and Johns Hopkins School of Medicine, Baltimore, directed by Dr. Helen B. Taussig.

The heart and great vessels in 1955, December 5-10, at Massachusetts General Hospital, Boston, directed by Dr. Paul D. White.

Recent advances in cardiovascular disease, January 16-20, 1956, at Southwestern Medical School of the University of Texas, Dallas, co-directed by Drs. Carleton B. Chapman and Maurice M. Scurry.

All registrations must be made through the Executive Offices of the College, E. R. Loveland, Executive Secretary, 4200 Pine Street, Philadelphia 4.

HENRY FORD HOSPITAL AND EDSEL B. FORD INSTITUTE FOR MEDICAL RESEARCH, DETROIT: International symposium on enzymes, units of biologic structure and function, November 1-3. For further information write to Dr. Clarence E. Rupe, secretary, International Symposium on Enzymes, Henry Ford Hospital, Detroit 2.

UNIVERSITY OF MINNESOTA, MINNEAPOLIS: Continuation course in neuroradiology for radiologists, November 7-12, at the Center for Continuation Study, under the direction of Drs. Harold O. Peterson and Leo G. Rigler.

UNIVERSITY OF MINNESOTA, MINNEAPOLIS: Continuation course in fractures for general physicians, November 21-23, at the Center for Continuation Study, under the direction of Dr. Wallace H. Cole, Professor, Department of Surgery, and Director, Division of Orthopedic Surgery.

Prolonged Labor

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M. EDWARD DAVIS

It is difficult to define the entity of prolonged labor by indicating arbitrary periods of time, for many factors enter into the management of the woman in whom labor continues beyond the average length of time. The length of labor in itself is only a measure of the scope of the problem. Most textbooks define a prolonged labor as one lasting more than 36 or 48 hours. However, today even 24 hours represents a long labor and the physician should be alerted to the possibility of trouble long before this period of time elapses.

It is not possible to indicate the time of onset of labor in every instance. A fair number of patients enter hospitals long before active labor has begun. There is a premonitory stage of labor which may begin during the last weeks or days before the end of the pregnancy. This stage is manifested by periods of irregular contractions which may last for a short or a long period, finally subsiding, then recurring. These episodes of uterine contractions are a necessary prelude to active labor for they result in cervical effacement, softening and even some dilatation. Such preparatory changes prognosticate a normal labor. Obviously some women enter the labor room in this premonitory stage and then spend many restless hours awaiting the onset of active labor. These women often receive barbiturates and analgesics which may upset the orderly mechanism of labor and precipitate abnormal uterine activity. Active labor begins with the onset of regular, rhythmic uterine contractions associated with progress in the form of continued cervical effacement and dilatation.

Incidence

The incidence of prolonged labor varies widely depending on the interpretation of the above factors. It may occur no oftener than in 1 per cent of all labors and has been reported in as high as 7 per cent. It is noteworthy that the intensive program of lay education for childbirth as well as the physiologic approach to the management of the woman in labor have decreased tremendously the incidence of prolonged labor at The Chicago Lying-in Hospital and most other institutions. Pregnancy and labor were regarded as pathologic states largely as a result of the high incidence of complications and the outrageous maternal and fetal mortality. There was an increasing tendency to make parturition more

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Presented before the thirty-ninth annual Assembly of the Interstate Postgraduate Medical Association at Minneapolis.

and more an artificial process using our growing knowledge in treating and preventing disease to safeguard our mothers and babies. The advent of antibiotics, the ease of blood transfusion, better anesthesia and all the brilliant scientific advances have decreased maternal mortality to such a low figure that it has been possible to alter the entire pattern of maternity care. Thus, the more physiologic pregnancy and labor have become, the fewer women have exhibited excessively long labors.

Preparation for Labor

Modern maternity care should provide an educational program for the young woman who is about to become a mother. This lay education should continue throughout the prenatal period and should embrace all teaching aids which are designed to indoctrinate the patient with the idea that labor is a normal physiologic process. Read has promulgated the thesis that ignorance engenders fear; fear causes spasm; spasm produces pain. The enlightened, well-prepared patient enters labor fully cognizant of what it involves. The early uterine contractions she tolerates without complaint. As the contractions become more uncomfortable she may ask for and receive some form of analgesia, not sufficient to obliterate her ability to participate in the most exciting drama in her life and yet sufficient to remove some of the discomfort. She is taught to relax and rest between uterine contractions, thereby conserving her energies for the second stage of labor. She will cooperate actively and effectively during this second stage. The termination of labor can be brought about spontaneously or with the aid of an episiotomy and outlet forceps delivery. Thus, one can attain the advantages of this physiologic approach as well as the modern methods of shortening the second stage and conserving the integrity and function of the perineum.

It is extremely important that the general practitioner who delivers babies be conversant with the present pattern of maternity care. Additional hours spent in indoctrinating parents for childbirth will be amply repaid in fewer complications, shorter labors and happier and better-adjusted mothers and fathers. Too few casual obstetricians have kept themselves informed concerning the present pattern of maternity care and its rich rewards.

Etiology

The most common factors which cause prolonged labor are cephalopelvic disproportion, an abnormal uterine mechanism and abnormal presentation. Excessively long labors are seen most often in primigravidas, and it is in these women that the pelvic size has not been evaluated by previous births. The grossly abnormal pelvis and the small pelvis can be discovered during the prenatal period. A careful clinical examination usually determines pelvic capacity. The few women with abnormal pelves should have x-ray pelvimetry.

Many factors have been listed as responsible for uterine inertia but in many instances no specific cause can be ascertained. The most consistent factor is that the vast majority of patients are in their first labors. Much importance has been attached to age and the elderly primigravida has always been regarded with concern. It is doubtful that the woman who has her first baby in her late 30s is less likely to exhibit a normal labor mechanism. However, if labor is excessively prolonged and progress is slow, an elderly primigravida deserves more consideration than a younger woman, for the importance of a living baby increases as the end of the reproductive period is approached. The present pregnancy may represent the last chance for the older woman to have a child.

Premature rupture of the membranes at a time when the premonitory phase of labor is incomplete and the cervix is long, closed and uneffaced interposes a long latent period before active labor begins. The irregular, arrhythmic contractions characteristic of this phase may keep the patient awake and uncomfortable and force the attendant to administer sedatives. This in turn may interfere with the normal labor mechanism and induce uterine inertia. Rupture of the membranes at the onset of active labor will rarely interfere with a normal labor.

Overdistention of the uterus because of excessive amniotic fluid or multiple preg-

nancy has been indicted as a cause of uterine inertia. If it is an important factor the rupture of the membranes will result in a reduction in the size of the uterine cavity and a normal labor mechanism.

Breech presentation is occasionally associated with uterine inertia. Here the breech supposedly provides an ineffective presenting part so that effacement is incomplete and dilatation is slow. However, in most breech presentations a normal labor mechanism is present and labor is not excessively long.

Calkins, who has studied the normal and abnormal labor mechanisms extensively, concludes that it is unlikely that there is an anatomic factor in the cervix responsible for its failure to dilate properly. There are probably physiologic causes for the so-called rigid cervix and the cervix that is responsible for excessively long labors.

Abnormal fetal presentation and failure of the occiput to rotate to an anterior quadrant are important causes of prolonged labor. Deflections of the head, such as brow and face presentations, present unfavorable diameters to the pelvic planes and increase the work load for the uterus. If the pelvic cavity is sufficiently large, the mechanism favorable, and uterine activity normal, such cephalopelvic obstacles can be overcome. However, if progress is slow, the labor mechanism ineffective, or the baby excessively large, the present tendency is to resort to an abdominal delivery. A carefully conducted trial labor will evaluate all of these factors better than clinical impressions arrived at by repeated examinations.

The most common predisposing factor in long labor is the failure of rotation of the occiput to an anterior position. The presenting part may be in the occipitoposterior position or it may rotate posteriorly as the labor continues. It may remain in the transverse position, resulting in a high transverse arrest of the head at the midplane of the pelvis or a deep transverse arrest with the head on the pelvic floor. Such malpositions are the daily problems of the active obstetrician. Fortunately, in many instances the head will rotate anteriorly if the contractions are good, or more rarely it may deliver as a persistent posterior. With recognition of these malpositions it is often possible to rupture the membranes with complete or almost complete dilatation, whereupon the head will rotate anteriorly. This often happens in the multigravida who seems to have reached an impasse. A few good contractions may result in rapid delivery. In many instances labor must be terminated when dilatation of the cervix is complete by manual rotation of the head and forceps delivery or by gentle rotation of the head with forceps, the so-called key-in-lock maneuver of DeLee.

Management

Much can be done to decrease the incidence of prolonged labor by meticulous prenatal care, which must include proper evaluation of the patient, good preparation for labor and intelligent conduct of active labor. Induced labor should be restricted to those patients who have medical indications for this procedure and in whom conditions for induction are present. The two common procedures for induction are rupture of the membranes and intravenous administration of pituitary extract. Sometimes these methods are combined. However, attempts to induce labor in a woman in whom the cervix is not "ripe" for induction—that is, it is almost completely effaced, short, soft and patulous—may lead to a long and difficult labor.

The size and normalcy of the pelvis can be determined with great accuracy. However, it is the size of the baby that must come through the pelvis which will determine the length of labor and its complications. Although it is possible to measure the various diameters of the fetal head rather accurately, a trial labor not only will evaluate cephalopelvic relationship, but, even more important, it also will provide an opportunity to test the labor mechanism. Thus, many patients must be allowed to go into labor and be observed carefully to determine if delivery will succeed without undue trauma. A test of labor will be the final arbiter as to how the delivery can be managed with greatest safety for both the mother and baby.

Trial Labor

A great many women enter labor rooms without positive findings that the pelvis is sufficiently roomy for the birth of the baby. In some the pelvic size is smaller than average; in others the inlet is somewhat flattened either as a late result of rickets or without obvious cause; in still others the baby is larger than average as determined by various means. The test of labor is a clinical means of evaluating the extent of cephalopelvic disproportion in such a patient.

In the "good old days" the test of labor lasted long enough to attain complete cervical dilatation, and a sufficiently long second stage was allowed to demonstrate beyond a doubt that the head would not progress down the pelvic cavity. Such an approach to the problem of the patient with the presenting part incompletely engaged was justified even though it resulted in a fetal loss as high as 25 per cent and fetal and maternal trauma which could never be evaluated properly.

Trial labor today should consist of a much more temperate evaluation of pelvic adequacy. In addition it is an excellent measure of the efficiency of the labor mechanism which in most instances will determine the success or failure of the trial labor. Thus, the woman who enters labor with the head of the baby unengaged or dipping into the pelvic inlet should be subjected to a very strict regimen. First, a careful study of the pelvic adequacy in the light of the size and presentation of the baby should be made. If gross disproportion is encountered because of pelvic size, an unfavorable fetal presentation or a baby who is much larger than average, elective cesarean section should be considered. However, in most patients we are dealing with a pelvis that is borderline in roominess and perhaps a baby that is somewhat larger than normal. An adequate trial of labor lasting from 6 to 18 hours, rarely even 24 hours, will decide the degree of disproportion between passenger and passages, and what is equally important it will demonstrate the effectiveness of the uterine mechanism. Labor contractions which are of good quality and conform to a normal pattern will soon overcome the moderate disproportion between the presenting part and the pelvis; the head will enter the pelvis, and labor will progress rapidly and eventuate in a safe delivery through the birth canal. The patient, who is usually a primigravida, will be spared an abdominal delivery which would have converted her entire reproductive function from a normal to a pathologic one. At The Chicago Lying-in Hospital there is increasing use of trial labor, and it is noteworthy that four of every five patients subjected to it deliver through the birth canal. The fifth individual is subjected to a timely cesarean section without increased hazard to mother or baby.

Certain safeguards in the conduct of a trial labor will decrease its hazards. The patient should be allowed to go into labor spontaneously, for an induced labor is never an entirely normal labor. The administration of pituitary extract during a trial labor is fraught with dangers far greater than the good it can accomplish. In spite of improved methods of administration it has no place here. Care should be taken to restrict orificial examinations in order to reduce the likelihood of infection. However, it is important to do a careful vaginal examination at the end of the trial period in order to evaluate properly the state of the cervix, fetal position and presentation and the degree of engagement of the presenting part. It is at this examination that the decision is made to continue labor or to resort to abdominal delivery. Favorable progress in the form of cervical effacement and dilatation, the descent of the head beyond the inlet and the demonstration that the lower half of the pelvis is adequate are the criteria for continuing the labor. A poor labor mechanism with irregular, desultory and ineffective contractions, the lack of complete effacement and progressive dilatation, and the failure of the head to engage in the inlet are evidences of a "failed" trial labor.

It is important to keep in mind that a test of labor can be applied only to the patient with a cephalic presentation. It is a clinical means of evaluating the adequacy of the inlet of the pelvis. Midplane and outlet contrac-

tions are far less common than inlet contractions, and these can be evaluated satisfactorily only by a careful pelvic examination. Gross inadequacies of the lower half of the pelvis should be confirmed by x-ray pelvimetry and given serious consideration at the onset of labor. Furthermore, a breech presentation in a borderline pelvis deserves even more serious consideration. There is no way to judge how the aftercoming head will fare as it proceeds through the inlet or midplane. With this type of case a more liberal use of cesarean section may spare irreparable fetal injuries and fetal death.

The treatment of uterine inertia has made some progress of late. Untimely sedation may seriously disturb the labor mechanism. Sedation administered at the optimum time will result in rest and better relaxation of the patient, and the contractions may become more effective. Such ideal results are seen in the patient who is making fairly good progress but who begins to weary because the work load is greater than normal. The baby may be excessively large or the head fails to rotate anteriorly but remains in the posterior or becomes arrested in the transverse. Such malpositions may add many additional hours to the labor and eventuate in difficult deliveries.

Uterine activity is the most important factor in prolonged labor. Effective uterine contractions can overcome many obstacles which contribute to long labors. In recent years there has been much study concerning uterine action and much fundamental knowledge has developed. Normal labor is typified by regular, rhythmic contractions entirely co-ordinated and following a definite pattern. This orderly activity results in cervical effacement and dilatation and ultimate descent of the head.

The use of oxytocic drugs to stimulate uterine activity has been resurrected and is gaining in popularity. The important new contribution is a method for the slow intravenous administration of minute amounts of pituitary extract. Although pituitary extract has lost none of the potent powers for damage which were responsible for outlawing its use in the treatment of uterine inertia, its force has been tamed by the intravenous drip. If it is used to improve uterine activity certain safeguards must be exercised.

PITOCIN® should be used rather than pituitary extract. Only a sufficient amount should be administered to stimulate normal labor. The physician should remain in constant attendance and the drip discontinued at any sign of abnormal uterine tonicity or excessive irritability. The contraindications to its use are even more important and include cephalopelvic disproportion, a uterine scar as a result of a previous cesarean section or myomectomy, uterine overdistention, parity of five or more, abnormal bleeding, or an elderly primigravida.

The patient who has been in labor for 12 hours and has failed to progress should have a pelvic examination to re-evaluate all the factors which may enter into her prolonged labor. The state of the cervix should be noted and the position of the head in the pelvis determined. If the cervix is partially dilated and the head well engaged, simple rupture of the membranes may result in more effective uterine contractions and a resumption of progress. It is not possible to favor anterior rotation of the head by the various manipulations which have been suggested, for only the labor mechanism can accomplish this.

In some patients with a poor labor mechanism the cervix dilates to 6 or 7 cm. and the head often is in posterior position and well down in the pelvis but no further progress occurs. Continued labor results in increasing edema of the cervix with a compensatory decrease in dilatation. Two modes of treatment can be used to terminate the labor, cesarean section or Dührssen's incisions. In our clinic we prefer the latter in the absence of any disproportion. The cervix can be incised at localities equivalent to 10, 2 and 6 on a clock face. The length of the incisions should be sufficient to allow for easy passage of the head. They can be repaired by interrupted sutures and healing is usually uneventful. We have reviewed a large series of patients who have had cervical incisions in their first labors and found that their subsequent labors were uneventful. Thus, Dührssen's incisions have a real advantage over cesarean section.

Four Vitamins in Ophthalmology

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FOR centuries man has subsisted on a score or more species of mammals, fish and plants. During this period, he has gained much knowledge about the use of such foods which has been to his advantage, but only in the last half century has he learned what an impact on his health the improper understanding of what is now known as the vitamin content of these foods has had, and nowhere has this been felt more keenly than in the field of ophthalmology.

My attention was first called to the importance of undernutrition in ophthalmology when I was studying under Dr. Ernest Fuchs in Vienna. At that time, no better place to study the effects of poor nutrition could be found than at the great government hospital at Vienna. In the provinces of Bosnia and Herzegovina, which then were under Austrian domination, the food deficiency was greater than anywhere else in Europe. The troops of both of these provinces were then being trained in Vienna. The oppressed people of the provinces had since 1875 been almost entirely isolated from the world, and for over a quarter of a century had been so beset with underground intrigue and aborted revolutions that they had given almost no attention to either commerce or tilling the soil. Thus, with their food supply almost cut off, the recruits from these provinces who were seen in the hospitals were very poorly nourished. Many of them gravitated into the eye wards, and were allotted to students for examination and case reports.

Hemeralopia and keratomalacia often were observed in these soldiers, but xerophthalmia was predominant. Accompanying this condition, a studding of the cornea with newly formed capillaries was sometimes seen. This was usually accompanied by photophobia, conjunctival irritation and blepharitis marginalis. Frequently, cheilosis also was present at the corners of the mouth. The patients aggravated this by irritation with soiled fingers. Others had red tongues or even pronounced glossitis. Although the tongues may not have been coated, the clinical picture pointed to pellagra, which was found more often then than now in poorly nourished persons. Incipient keratomalacia was seen frequently and was associated with a condition of the conjunctiva which is now known as Bitot's spots. These usually were observed to clear up in a few weeks, or at least to be improved by the administration of cod-liver oil or chopped

liver slightly cooked. It was impressed on us as students that we should look nowhere else for the cause of hemeralopia, keratomalacia and xerophthalmia than in disturbances of nutrition, and this was proved to the satisfaction of the students by their observation that these conditions improved and sometimes disappeared under the influence of good food, which included cooked liver, and with the administration of cod-liver oil.

Fuchs taught his students and has stated in his book[1] that perinuclear cataract occurred in childhood as the result of rickets, and he quoted Homer to the effect that rachitic interference with nutrition affects structures, "especially the teeth and the crystalline lens." He continuously emphasized that the intake of more fluid in cases of malnutrition was necessary, and he stated in his book that "it is said that in diabetic cataracts which have not advanced too far a partial disappearance of the opaqueness has sometimes been observed after a successful treatment of the diabetes by the Carlsbad water cure."

In checking on our reports, we were told that keratomalacia developed almost wholly in consequence of enfeebling nutritional influences, and our attention was called to the fact that the disease also occurs frequently in Russia "as there it attacks infants during and after the time of the great fast of Easter" because during that period mothers lose their milk in consequence of fasting. It was emphasized that the chief factor in successful treatment of keratomalacia was an attempt "to support the patients' strength by means of bettering their nourishment."[1]

It is noteworthy that other authors of this period, when little was supposed to be known about human nutrition, were not insensible to the fact that such eye diseases as hemeralopia, xerophthalmia and keratomalacia were food deficiency diseases. De Schweinitz[2] stated that keratomalacia responds to constitutional treatment, and he suggested cod-liver oil as the remedial agent of choice. He also attributed night blindness to lack of nutrition, and recommended among other restoratives the use of cod-liver oil. In 1897, Swanzy[3] classified diabetic cataract as a disease due to disturbed nutrition, and he, as did Fuchs, mentioned that great and prolonged fasts had for years been advocated for diabetic patients to the great disadvantage to their vision.

That these older authors were correct in this conclusion, although knowing little or nothing about vitamins, was later mentioned by Foster,[4] who recognized keratomalacia as a food deficiency disease and suggested vitamin A as found in carotene or in liver oil as the most outstanding remedy for the disease. Foster, in discussing deficiency diseases, also listed under pellagra the occurrence of cataract in man and in rats. As man cannot be subjected to prolonged experimental starvation to prove the cause of any disease, this cannot be made to mean that cataract does not occur in man as a nutritional condition, and one may note that Fuchs[1] classified cataract as a complication of pellagra.

Malnutrition in the Southwest

During the early years of my practice of ophthalmology, I had the opportunity of observing firsthand many of the ill effects of malnutrition. At that time, I derived a large part of my practice from among the share-croppers of the eastern section of Texas. Among these patients, poor nutrition was the rule rather than the exception. Many of them were victims of pellagra, which now is rarely seen in its severe form. Their chief diet consisted of overmilled corn bread. In many of my patients' families there was no milk and, consequently, no butter. The substitute for butter in almost all instances was cane molasses. Water was provided almost wholly from surface wells. It was tasteless, and most tenants drank it only in the event of extreme thirst. There were few eggs to be had because these people were migratory, moving from one place to another in the hope of bettering their lot. They therefore had no disposition to acquire and carry with them laying hens. All this had a disastrous effect on their eyes.

Many of the patients from this area had keratinized epithelium of the cornea, and the cornea was wrinkled and steamy. Elevations on the ocular conjunctiva often were present and had a frothy appearance. I had attempted

in similar conditions in Vienna to brush these elevations off, and I repeated the effort here, but they could not be removed in this manner. These raised surfaces on the conjunctiva were apparently greasy, for when solutions were instilled into the eyes they failed to cover the elevations but swept around them. These elevations, of course, were the now well-known Bitot's spots. Although Fuchs[1] had already described the condition, he did not give these areas a special name, and Bitot's spots did not become a part of the terminology of ophthalmology until sometime later. Among the patients with pellagra seen at this time were a number with eye manifestations, such as chronic blepharoconjunctivitis, and there sometimes was an associated chronic hyperkeratosis which extended outward from the nose and eyelids. This condition was then described as the pellagra mask.

At this time, incipient and mature cataracts were observed among these patients from Eastern Texas who worked long hours in the summer sun, and I began to suspect that the development of the cataracts was influenced in some cases by a small intake of fluids. Whether or not cataract is influenced by dehydration of the crystalline lens is a moot question, but it has long been believed by many authors that persons living in hot climates should drink more than the average amount of water. That the great loss of water occasioned by sweat in hot weather is dehydrating has recently been proved by studies made at the Harvard Fatigue Laboratory[5] where it was found that a man doing hard labor in a heated atmosphere loses through his sweat glands a quart of water every hour.

Because of the improvement of foods due to education and to commercial advertising of vitamins, advanced pellagra is now seldom seen in patients from Eastern Texas. However, Simpson[6] described a condition which he termed secondary pellagra; that is, pellagra symptoms which have occurred after gastrointestinal complaints of long standing in cases in which the diet list had of necessity become very meager. Cures of many of these were said to have been brought about by giving foods rich in vitamin B_2. Hutchison and Paterson[7] have described a modified type of pellagra which occurred in the asylums and orphanages of England. Ocular symptoms occurred in many of the cases. In some of these institutions, the diet provided for inmates was corrected so as to increase the amount of protein foods with the result that 40 per cent of the inmates, including the patients who had ocular symptoms, recovered immediately, and others were reported to have made distinct gains.

It is from this type of so-called pellagra occurring in persons otherwise not in good health and whose diet is reduced in vitamin-bearing foods and who have occasional attacks of dermatitis of the eyelids, blepharoconjunctivitis and, occasionally, incipient cataract that the ophthalmologist draws much of his work in certain areas of the Southwest. While the former use of corn meal which had been robbed of its vitamin content in the process of milling has largely disappeared because of education, only a meager amount of butter, eggs and legumes are added to the diet in the majority of instances.

Whether the absence of vitamin B_2 in the diet is the sole cause of pellagra or whether the condition may be largely attributable to a tryptophan deficiency, as suggested by Wohl and Waife,[5] is a matter still under discussion.

In view of this brief appraisal of dietary needs as seen in the practice of ophthalmology, and since the subject is entirely too broad to consider fully in a brief article, I shall limit this discussion to consideration of the various merits of vitamins A, C and D and B_1 and B_2.

Vitamin A

Rea[8] said that the importance of vitamin A in the production of vision was first discovered in Denmark in 1917. At that time, most of the butter of Denmark was being shipped to Germany where it brought a very high price, and oleomargarine was substituted for butter. Many of the children of Denmark became affected by xerophthalmia. As soon as possible, butter was provided for these children, with the fortunate result that all of them recovered. During this emergency, Møllgaard[9] of Denmark found that the addition of vitamins and butter to the oleomargarine did a great deal

to relieve the symptoms which were brought on by the absence of vitamin A.

Vitamin A is thought to stand second as a restorative agent in the practice of ophthalmology. As chlorophyll in plants is due entirely to the effect of sunlight, so almost to an equal degree is vitamin A a product of the sun. In all mammals, birds, reptiles and fish, by a process little understood, vitamin A is stored in the liver. Much of it reaches mankind through the lower animals, as in milk and butter and in eggs and fat. Fish muscle contains a small amount of this vitamin, but fish liver (cod-liver oil) is its chief source for therapeutic use. It is also present in the livers of all animals and birds used for human consumption. Human beings can live for long periods without taking into the body vitamin A, for, in human beings, as in all animals, it is stored in the liver to be used only when needed.

Alpha, beta and gamma carotene, which when combined are generally spoken of as carotene, are found in a large percentage of foods known to have a provitamin A activity. They are all converted into vitamin A in the liver where they remain until their utilization by the body is necessary. The richest sources of this vitamin are to be found in liver oils such as salmon, cod and halibut. Combined with vitamin D, vitamin A occurs in plants and fruits which receive much sunlight during growth. It is also found in milk and milk products as well as in such vegetables as carrots and turnips and in leafy vegetables such as spinach, broccoli, beet tops, chard and mustard greens.

It is well known that absence of vitamin A (carotene) leads to atrophy of epithelial structures. When present, this is the sign usually first seen by the ophthalmologist at the beginning of his examination. A keratinization of the ocular conjunctiva gives this structure a wrinkled appearance. If avitaminosis is pronounced and of long standing, the cornea may be involved, and keratomalacia may be present. This is now seldom observed, but mild ocular manifestations of vitamin A deficiency, such as thickened areas on the conjunctiva, are usually present.

Absence of vitamin A is likely to be ushered in by an attack of night blindness, which gradually progresses in severity. This nearly always precedes any of the observable ocular lesions, and may be more or less constant for days or weeks before external ocular manifestations become evident. Continued lack of vitamin A in the diet leads in many instances to keratomalacia or to retrobulbar neuritis which may partially or wholly destroy the patient's vision.

Some authors consider night blindness to be the chief, if not the sole, result of an absence of vitamin A in the diet. Other authorities, however, believe most night blindness is hereditary and due to retinitis pigmentosa. This, in turn, is not due to vitamin deficiency, nor can the condition be improved by the administration of vitamins of any kind in any amount. This conclusion was derived from studies of prisoners of war during World War II and the Korean conflict. Foster[4] said that night blindness comes about by a destruction of normal visual purple in the retina, and he has found that its relief is greatly augmented by wearing sunglasses during the day. Wood,[10] while attributing most instances of night blindness to lack of vitamin A, did not go so far as to think that it is the sole cause but suggested as the most feasible treatment vitamin A preferably supplied by large doses of cod-liver oil. This appears to me to be somewhat of an admission that the lack of vitamin A is the chief, if not the only, cause of night blindness.

Solares,[11] after observing Bitot's spots in several cases of night blindness with xerosis of the conjunctiva, said that all of the patients improved and that a number of them recovered completely after having been given foods rich in vitamins A and D. Rea[8] recounted how an Indian student in London, seeking to maintain himself on the usual Indian diet of his caste, had keratinization of the corneal epithelium which cleared up slowly after he adopted a diet containing more than an average quantity of vitamin A, such as milk, butter, eggs and vegetables, in addition to his usual Indian diet.

A wise provision of nature lies in the fact that carotene is a substance so constant in

plant life that the intake of the plants which contain this substance usually is sufficient to avoid a deficiency of vitamin A. Further than this, carotene, when not in use, is put in liver storage as vitamin A until utilized. Other basic sources, when more vitamin A is needed, are liver oil, chiefly cod-liver oil, and milk and milk products, such as butter and cheese.

Vitamin C (Ascorbic Acid)

This is one of the most serviceable vitamins used in ophthalmology. It is, however, more unstable in constant value than any of the vitamins because of its tendency to rapid deterioration. After being harvested and exposed to sunlight or even to the atmosphere, vegetables and fruits which all contain ascorbic acid are subject to a rapid loss of their vitamin content. The chief exception to this lies in the families of leafy vegetables which retain vitamin C longer than other fruits and vegetables when kept in a cool place.

Vitamin C is such a well-recognized content of normal food that the public is now rather well acquainted with it. It occurs in fruits such as oranges, grapefruit, bananas, apples and lemons. In lesser quantities, it is found in milk and milk products. In vegetables, such as potatoes, it is present in moderate quantities. Much of the vitamin content of vegetables is lost by the cooking process, because vitamin C, being water-soluble, escapes in the water in which such vegetables are boiled.

It has lately been discovered that all berries and berrylike fruits are rich in vitamin C. This is especially true of a small pulpy fruit resembling a berry which the School of Tropical Medicine of the University of Puerto Rico recently found to be richer in vitamin C than any other known substance. This berrylike fruit, the acerola, is about to be added to a list of fruits suitable for use in deficiency diseases by major growers and packers, and it is their intention to add acerola juice to the juice of other fruits rich in vitamin C for use as a protective food for patients suffering from malnutrition.

All fruits, when canned immediately after being harvested and before harmful deterioration takes place, and when not subjected to prolonged boiling, retain to a moderate degree the vitamin C content. It may be said that the vegetables sometimes used for canning have lost nearly all of their vitamin content by spoilage and may be considered almost inert in vitamin C. However, the process of canning tomatoes, it is claimed, does not interfere greatly with their potency of vitamin C, even when they are exposed for long periods to what would be considered ruinous atmospheric deterioration for other vegetables.

The study of vitamin C prompts some thought relative to the crystalline lens. Krause[12] said that vitamin C is present in the human lens, and in the vitreous and aqueous humor, but that no vitamin C is found in the aqueous humor after the lens is removed. The question naturally arises, does the normal vitamin C content of the aqueous humor safeguard against cataract? Josephson[13] suspected that it does, and he called attention to his findings that the administration of vitamin C reduced the opacity in cases in which cataracts had been caused by dinitrophenol and to a lesser degree in cases of other types of cataract.

In ophthalmology, the effect of the absence of vitamin C is often manifested in cases of cataract among elderly persons whose daily rations consist of cereals, such as corn bread and white bread with the addition of cooked vegetables, and who drink tea and coffee but drink little water. It is also known that some of these poorly nourished persons, as a result of the absence of normal intake of ascorbic acid and fluids, are subject to repeated attacks of hemorrhagic retinitis, and that subconjunctival hemorrhages are not infrequently seen among them. While some of these patients have in their diet a modicum of milk, this is usually pasteurized. It is known that vitamin C in milk is either lessened or destroyed by boiling, depending on the length of time consumed in the process. For this reason, some pediatricians refuse to use pasteurized milk if known pure whole milk can be obtained.

Vitamin D

From the viewpoint of ophthalmology, the absence of vitamin D as seen in rickets is of

more than the average importance. Langdon-Brown[14] said that the association of cataract with rickets and malformation of the teeth is so well known that the fact is rarely discredited, and he remarked that one feature in patients thus affected is predominant, namely, that hypocalcemia is usually associated with the cataract.

Von Bahr[15] claimed that the absence of vitamin D causes cataract in rats, and he also stated that cataract sometimes accompanies rickets if tetany supervenes. Rea[8] said that improper feeding can be the cause of zonular cataract in human beings and that the fault may be repeated in the children of the same family. He believed that this probably explains the familial incidence of such cataracts.

As a hint to the association of rickets in general medicine as well as in ophthalmology, Rea[8] quoted Osler as saying that night blindness and, occasionally, day blindness have been traced to anemia of the retina brought on by rickets. Rea also claimed that rickets, besides being a source of lamellar cataract, is also a fruitful source of myopia and follicular conjunctivitis.

Jeghers[16] said that rickets is often due to a lack of vitamin D brought about by an absence of sunlight from the body. This he believed is induced by window glass, smoky atmosphere or dense clouds. It has been noted that in Negro children with heavily pigmented skin, rickets, when once established, is a more resistant disease than it is in white children. This has been thought to be due to action of their pigment in shutting out sunlight.

The public is becoming conscious of the fact that vitamin D deficiency is the predominant cause of rickets. As a result, advanced rickets is now rarely seen because of the general education as to its cause and because of the activation with vitamin D of many commercially prepared foods by exposure to sunlight.

In medicine, vitamin D is the term usually applied to synthetic ergosterol, but its chief source is fish livers, preferably those of the cod. The average foods reaching our tables, with the exception of fish muscle, eggs and milk, contain little of this vitamin. It is, therefore, thought necessary in children to supplement vitamin D as found in foods by a sufficient amount of outdoor life with sunshine. The artificial activating of the food intake of children by ultraviolet radiation or direct sunlight is also believed advisable.

One very important factor in the treatment of rickets had its origin in the discovery that most of the vitamin D content of all grains lies in the covering or hull. For centuries, this health-promoting property was blown away to separate it from the flour being milled. This worked havoc on those who could afford this prized but depleted food. Such persons and their children paid the penalty for this in chronic malnutrition and its results, while the underprivileged subsisting on home-milled "black bread" thrived. This is reminiscent of the Southwest, for the absence of vitamin D did not become a menace here during the pioneer days of the mortar grinding of grain, when the hull of the staple food, corn (maize), was retained with the meal, but only after the water gristmills came into common use and the corn was then robbed of its hull along with its antirachitic vitamin D.

Vitamin B_1 and Vitamin B_2

Vitamins B_1 and B_2 are the antipellagra and the antiberi-beri vitamins. Thiamine is found only in vitamin B_1, and riboflavin is contained in vitamin B_2. Foods such as fish muscle and liver contain both vitamin B_1 and vitamin B_2. Milk and its products contain lesser amounts of these two vitamins. These vitamins are abundantly present in lean meat, green vegetables, and fruit slightly cooked, and in such legumes as peas and beans in the pod when not overcooked. Wheat germ, eggs and yeast provide rich sources of vitamins B_1 and B_2.

Day, Darby and Langston[17] classified riboflavin as a cataract-preventive factor, and have demonstrated that cataract developed in a large percentage of rats fed a diet deficient in riboflavin or vitamin B_2. Whether or not the same would apply to man is not shown, but these authors concluded that a rich riboflavin diet will prevent cataract in the rat. Commercially, vitamin B_2 represents the watery fraction of milk which is left after the manufac-

ture of cheese. What effect this would have on cheese as an article of diet was not made clear by these investigators.

Excessive deficiency of vitamin B_2 in the diet, ariboflavinosis, predisposes to corneal invasion by new capillaries. This can readily be distinguished from beginning pannus trachomatosus by anyone with experience in ophthalmology by noting the location, intensity and direction of newly formed corneal vessels. According to Rea,[8] the chief eye lesions associated with lack of vitamin B_2 as found in pellagra consist of cataract, toxemia of the optic nerve, and, occasionally, diplopia. The prevention of vitamin B_2 deficiency leading to the establishment of pellagra and its complications consists wholly of the administration of vitamin B_2 and foods rich in this vitamin.

In the paralytic form of beri-beri there is a paralysis of the external rectus muscles which causes internal squint.[18] In cases of this type of beri-beri, which often is accompanied by hemeralopia, the peripheral field of vision is contracted, and there is a dense central scotoma. If treated early, the patients improve, and some are cured by the administration of vitamin B_1. That a dearth of vitamin B is likely to affect the optic nerve has been suggested by Moore,[19] who reported a widespread occurrence of retrobulbar neuritis in Nigeria which was brought under control by the administration of foods rich in vitamin B.

Langdon-Brown[20] called attention to the fact that cataract in cases of rickets associated with malformation of the teeth and hypothyroidism is well known. Sabry[21] also said that "rickets is a menace not only by the occurrence of cataract but due to the effect of the toxin on the optic nerve." He believed that this is the source of the diplopia often seen in cases of rickets.

As everyone now knows, vitamin B_1 was the first vitamin studied after Eijkman's discovery that the sailors of the Japanese navy, living for the first time on unhulled barley, also for the first time in history escaped beriberi, whereas the officers with their accustomed diet of polished rice suffered from beriberi as they had before. Eijkman, taking this fact into account, immediately began experimenting with fowl, to some of which he fed polished rice exclusively. Polyneuritis, a condition akin to beri-beri in man, developed in the fowl. Others were fed unhulled rice and escaped polyneuritis. Eijkman then made public his discovery relative to these facts, but his findings were generally passed over until 1911 when Funk, who had done much work on foods, combined the two words *vita* and *amines*, left off the "s," and coined the word vitamin. While vitamin B_1, the specific for beri-beri, is not of such great importance in eye conditions as are vitamins A, B_2, C and D, the discovery made by this very modest observer has opened up a study of vitamins which has become more important to the human race than all the dietary advances which had been made previously.

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Radical Mastectomy Designed to Prevent Excessive Blood Loss and Postoperative Lymphedema of Arm

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DONALD GUTHRIE

ANY operation which is planned to reduce loss of body fluids, loss of heat, and loss of blood is worthwhile. The operation which I shall describe has for its primary purpose the control of the arterial supply to the pectoralis major and pectoralis minor muscles and to the breast itself. Later on I shall discuss a means of controlling or preventing the rare but distressing condition called lymphedema of the arm—lymphedema not due to metastasis but caused by a block in the superficial lymphatic drainage of the arm to chest wall.

At the Guthrie Clinic during the period of March 1, 1910 to October 1, 1954, a total of 979 patients underwent operation because of malignancy of the breast. Of these, 974 were female and five were male patients. These figures illustrate that carcinoma of the male breast is extremely rare. In all five cases, metastasis was rapid and all five male patients were dead within a year and a half.

The operation to be described attacks the arterial supply to the breast, first in the axilla. An incision known as the Rodman incision is made, which is kept off the arm (figure 1). The first step is to divide the tendon of the pectoralis major muscle where it is attached to the bicipital groove of the humerus (figure 2). Next the tendon of the pectoralis minor muscle is separated from the coracoid process of the scapula (figure 3). One step here is to be careful of the long thoracic artery and vein which are shown just above the border of the muscle. After this step, the axilla is exposed by dividing the axillary fascia, which is a very firm structure.

We strongly advise a careful, gentle, clean anatomic dissection of the axillary contents (figure 4), beginning at the top and dividing

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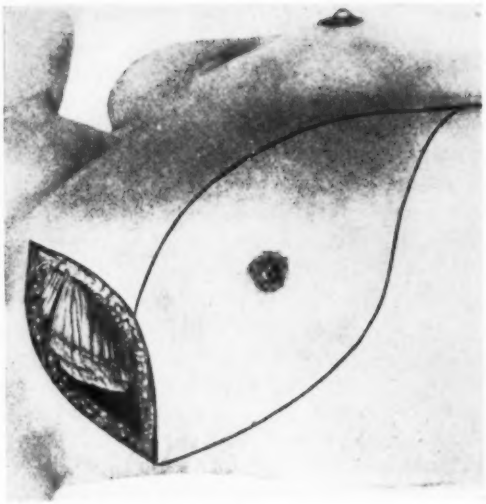


FIGURE 1. The Rodman incision.

FIGURE 2. Division of tendon of pectoralis major muscle.

the acromiothoracic artery and vein, next the alar thoracic (a small branch which is sometimes absent), then the long thoracic artery and vein, and finally the subscapular or posterior thoracic artery and vein. The respiratory nerve of Bell supplying the serratus magnus muscle and the posterior thoracic nerve to the latissimus dorsi muscle are identified and protected. Years ago, Halsted advised against rough dissection of the axillary space because the infections in the axilla postoperatively are of the low-grade type, hard to rec-

FIGURE 3. Division of the tendon of the pectoralis minor muscle from the coracoid process of the scapula.

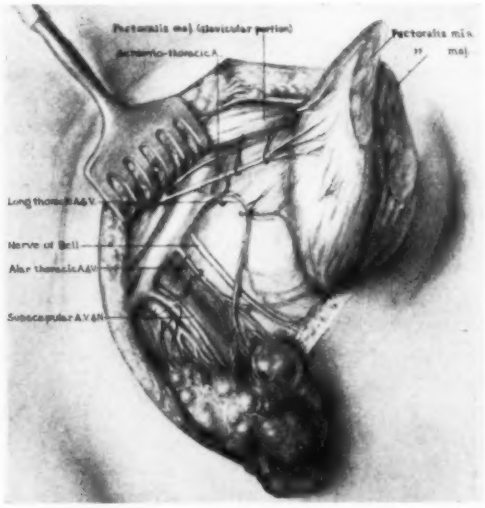


FIGURE 4. Anatomic dissection of axilla.

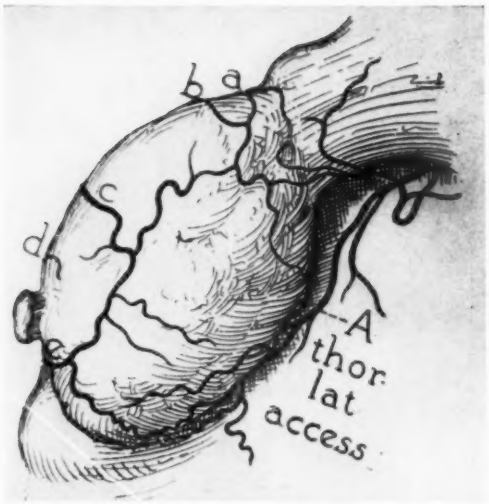


FIGURE 5. Blood supply to the breast. About 25 per cent is from the long thoracic artery.

ognize. We believe this is one of the factors in the production of postoperative lymphedema of the arm.

The Rodman incision, made an inch below the clavicle, two fingerbreadths mesially to the sulcus formed by the deltoid and the pectoralis major muscles, extends downward over the external border of the pectoralis major muscle.

Figure 5 shows the blood supply to the breast. About 25 per cent comes from the long thoracic artery, but its main blood supply is from the perforating branches of the internal mammary artery. It is estimated that 70 to 75 per cent of the blood supply of the breast comes from the second, third and fourth perforating branches of the internal mammary artery (figure 6).

The completed dissection is illustrated in figure 7; everything has been removed except the long respiratory nerve of Bell to the serratus magnus muscle and the subscapular nerve to the latissimus dorsi. If either of these nerves is sacrificed it is much more important to conserve the long respiratory nerve of Bell because if paralysis of the serratus magnus muscle occurs postoperatively, the patient will have a winged scapula, which is a hideous deformity and also it is very crippling.

Figure 8 shows the completed operation. We use very fine cotton for our ligatures. We close the skin with figure-of-eight sutures of crochet cotton, which affords a much more comfortable dressing postoperatively than silkworm gut. The dressings stick to the silkworm sutures and these sutures will often cut through the skin under tension, whereas the crochet cotton will not. We employ skin grafts in about 10 per cent of our cases.

The first dressing which is applied following radical mastectomy is very important (figure 9). The arm is extended well out from the body, a pressure pad is put on to obliterate dead space, and the arm is moved immediately postoperatively—another factor, we believe, in preventing postoperative edema of the arm. We employ drainage in very few of our cases.

The function of the arms after double mastectomy is demonstrated in figures 10 and 11.

Figure 12 illustrates an example of that rare, distressing, crippling and incapacitating condition of postoperative lymphedema of the arm, not due to metastasis. Let me make that point clear! Another example is shown in figure 13—a huge, swollen, edematous arm which cannot be pitted; such an arm may weigh 8 or 9 lb. more than the other arm.

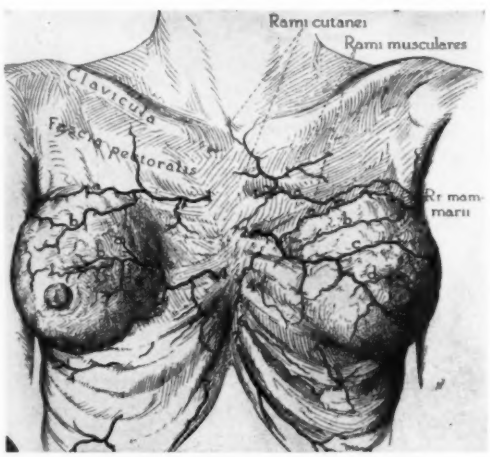


FIGURE 6. Main blood supply to the breast (75 per cent) is from the second, third and fourth perforating branches of the internal mammary artery.

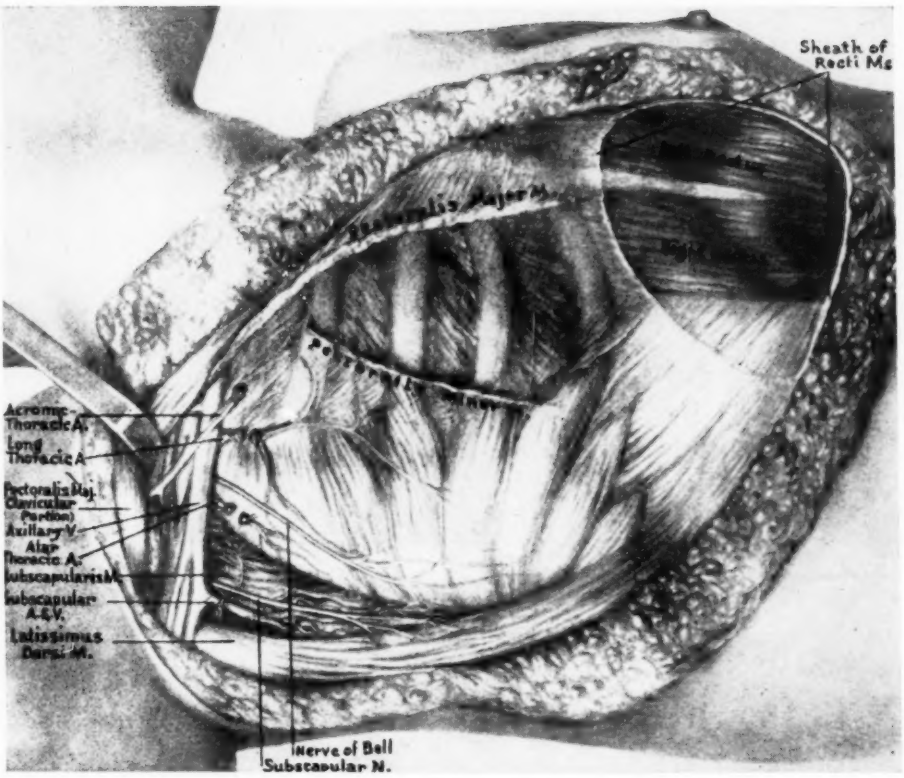


FIGURE 7. The completed dissection.

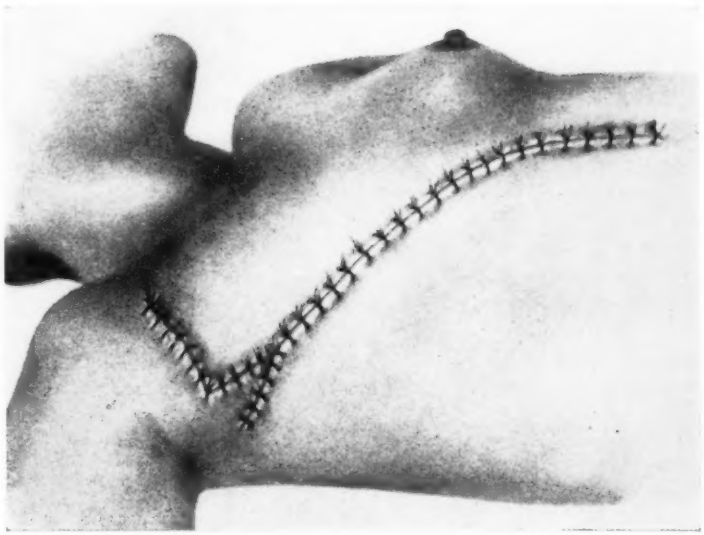
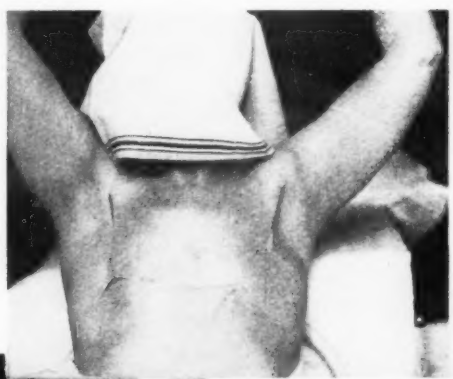
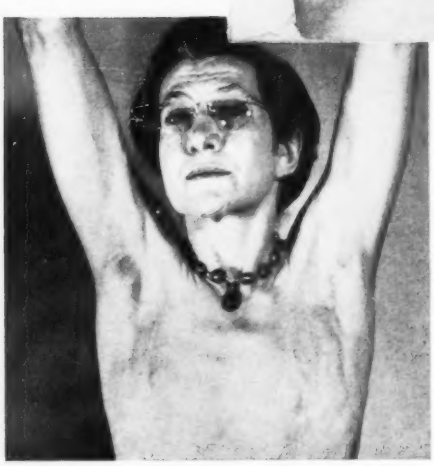


FIGURE 8. Wound closed.

FIGURE 9. First dressing after radical mastectomy.





FIGURES 10 and 11. Photographs of patients who underwent double radical mastectomy, demonstrating the function of the arms.

FIGURES 12 and 13. Examples of postoperative lymphedema of the arm *not* due to metastasis.

Another curious and distressing development in some of these cases is a cellulitis attended with mottling of the skin, high fever and chills. Its origin is still obscure but it is thought to be due to infection. We are taking cultures of the fluid but it is too early to report on the results.

As I have said, we like to have free motion of the arm; we like to keep the scar away from the arm. The incision of the Willy Meyer operation which, as far as priority is concerned, we call the Meyer-Rodman operation is shown in figure 14. Willy Meyer reported this operation at a meeting of the New York Surgical Society before Halsted reported his operation, which was published in the *Johns Hopkins Bulletin*. Willy Meyer advised the removal of both muscles, as did Halsted, and complete dissection of the axilla, but Willy Meyer dissected the axilla first, which we believe is a very valuable contribution.

In the Halsted operation, the incision extends well out in the arm, mobilizing the breast first before going into the axilla (figure 15). It is a good thing to inspect the axilla first rather than to do that as a last step in a mastectomy, because a few of these cases will prove to be inoperable because the glands are so adherent to the axillary bed.

Figure 16 illustrates the old Collins-Warren incision.

Notice in figure 17, which shows the Elsberg incision, that a large flap is transposed and covers that wide defect. Also note how far down in the arm the incision extends.

William Beck of our clinic suggested an operation which we have used to advantage in well-chosen cases. Its purpose is to connect the superficial lymphatic circulation of the arm with the chest wall. We select our cases with a great deal of care. We employ venography to make sure that the axillary vein is patent and study the case carefully to ascertain that metastases are not present. We are loath to accept for operation patients showing fibrotic changes in the axilla and the chest wall following deep irradiation. All patients have determinations of basal metabolic rate, albumin-globulin ratio and blood cholesterol. Stereoscopic plates of the chest, to exclude radiation fibrosis or metastasis, and a phlebogram of the axillary vein are made (table 1).

FIGURE 14.
Willy Meyer incision.

FIGURE 15.
Halsted incision.

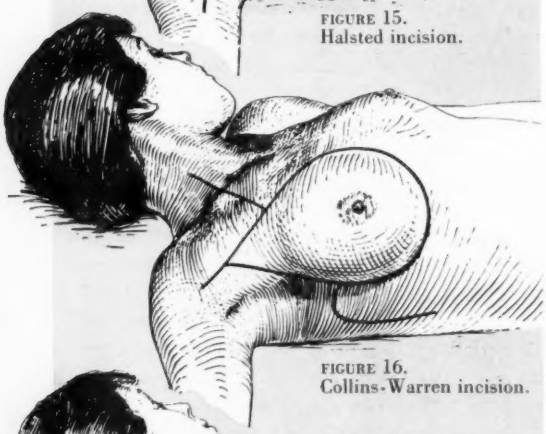


FIGURE 16.
Collins-Warren incision.

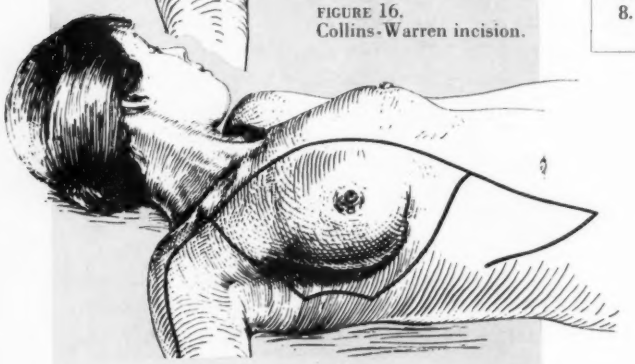


FIGURE 17.
Elsberg incision.

We prepare them for operation by giving thyroid extract and DIAMOX®, applying an elastic glove (converted stocking), infiltrating the tissues with WYDASE®, and elevating the arm on an improvised frame at night (table 2).

At operation, a small incision is made on

TABLE 1

PROCEDURES IN DIAGNOSIS OF LYMPHEDEMA

1. Basal metabolic rate
2. Albumin-globulin ratio
3. Blood cholesterol
4. Chest x-ray (radiation fibrosis or metastasis)
5. Phlebogram of axillary vein

TABLE 2

MANAGEMENT OF LYMPHEDEMA

1. Thyroid extract
2. Diamox
3. Elastic glove (converted stocking)
4. Tissue infiltration with Wydase
5. Elevation of arm on improvised frame at night
6. Implantation of celloidin strips over lymph barrier area

TABLE 3

OUTLINE OF PROCEDURE FOR IMPLANTING CELLOIDIN STRIPS

1. Three day preparation on airplane splint
2. Two day preparation with soap and water
3. Small incision on chest wall
4. Long forceps plunged through incision
5. Incision over point of forceps
6. Strip clasped and pulled back
7. Compression dressing for four to six weeks
8. Strips removed under local anesthesia

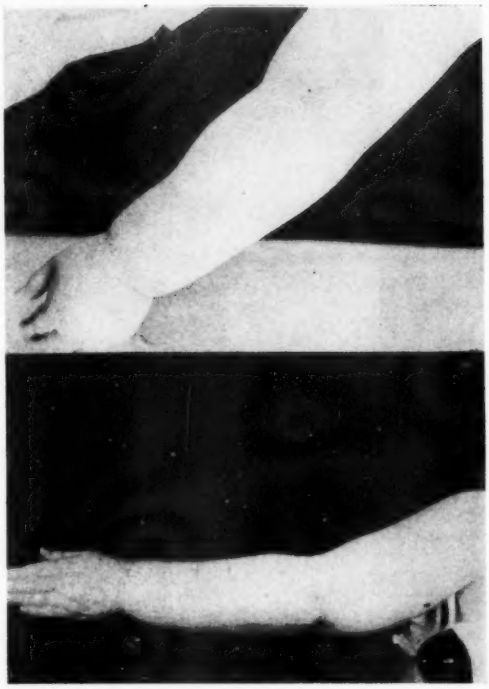


FIGURE 18. First operative case.

the chest wall. A long forceps is then plunged through the incision to a point on the arm. We incise over the point of the forceps, and the strip is grasped and pulled back, burying the ends beneath the skin. A compression dressing is applied for four to six weeks. The strips are removed under local anesthesia (table 3).

The first patient on whom I performed this operation was as surprised as I was with the excellent result (figure 18). She lived nine years, and I had the opportunity of seeing her many times. Her edema never recurred.

Figure 19 shows the good results obtained following operation on a doctor's wife. Preoperative and postoperative photographs of another doctor's wife are shown in figures 20 and 21. I get a great many doctors' wives to operate on, and while this is a compliment it is a heavy responsibility. Figures 22 and 23 are of another doctor's wife. Both she and the doctor are happy about the result. Figures 24 and 25 are of a doctor's sister, a teacher who was badly incapacitated by lymphedema of the arm.

This operation which Beck suggested and described is very simple and safe. It is all done on top of the muscles. We use three celloidin strips 1½ in. wide and 6 or 7 in. long. We make a small incision on the chest wall with a curved forceps on top of the muscle to the arm, make another small incision and bring the strips through, three in front and three in back.

One of the patients on whom I performed this operation had a very swollen right leg as a result of tuberculous hip disease which required many drainage operations (figures 26 and 27). Figures 28 and 29 illustrate the end result.

I believe this operation might have value in fracture cases where there is tremendous swelling of the legs following fractures of the thigh. The orthopedic department of the Guth-

FIGURE 19. Good result following operation.

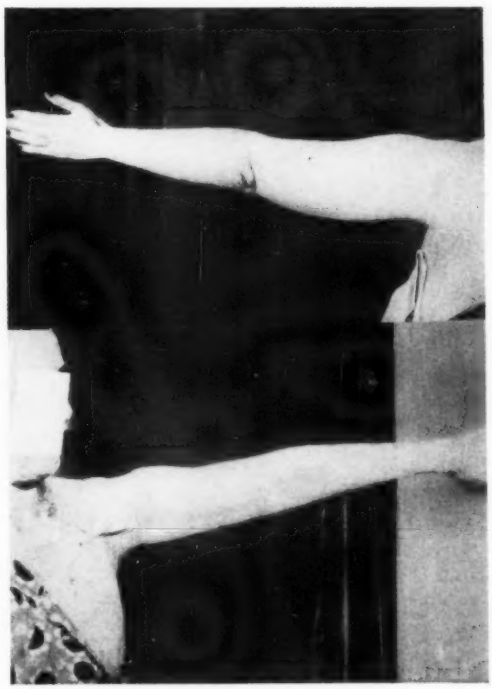


FIGURE 20. Preoperative photograph.

FIGURE 21. Same patient as in figure 20; good postoperative results.

FIGURE 22. Preoperative photograph.

FIGURE 23. Good results following operation on patient shown in figure 22.

FIGURE 24. This patient was severely incapacitated because of the lymphedema of the arm shown here.

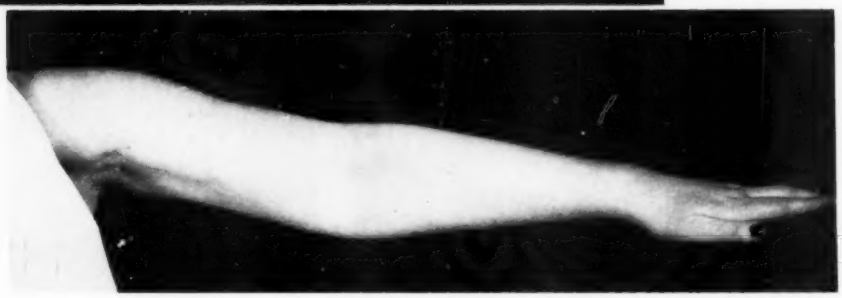
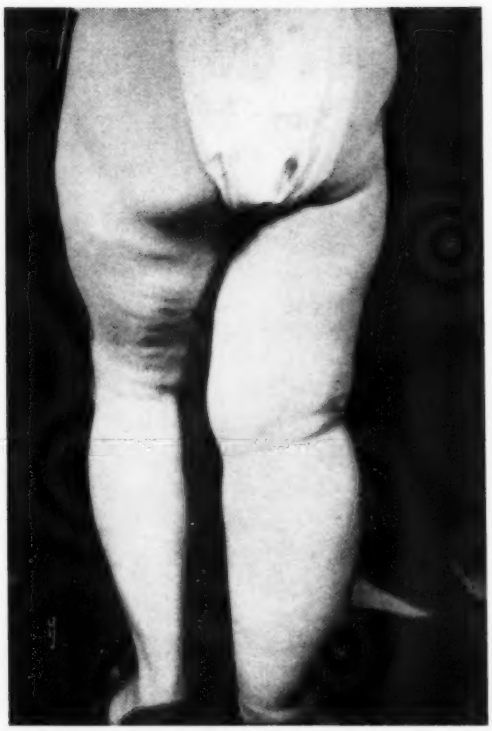


FIGURE 25. Same patient as in figure 24, after the operation.



FIGURES 26 and 27. Preoperative views of a patient with swollen right leg due to tuberculous hip disease.

FIGURES 28 and 29. End result in case of patient shown in figures 26 and 27.

rie Clinic is now beginning to investigate its use in such cases.

Longer celloidin strips are now available. The very long one is used for badly swollen hands. It is put under the skin and goes up the entire length of the arm over to the chest wall. Before we had these long strips we used crochet cotton and nylon, beginning at the wrist and going up until we got up to the upper part of the arm, and then we put these celloidin strips through.

We believe that low-grade axillary infections, incisions that extend down onto the arm, and long periods of immobilization of the arm are factors in the production of the distressing condition of postoperative lymphedema.

Hypoglycemia in Infancy and Childhood: Differential Diagnosis and Therapy

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My main purpose in presenting the patients I have here today and discussing their problems is to make it possible for you to recognize such cases and then treat them early. We have long heard a great deal about the desirability of early diagnosis and therapy in other disease states, but what I want to talk about today is, for most people, a new clinical entity in which early treatment is paramount. I put it so boldly because otherwise you are going to overlook the cases.

It is extremely important that patients with severe hypoglycemia be recognized, because the brain cells, with their peculiar metabolism, can use glucose only. In a young child whose brain is constantly growing and undergoing development, deprivation of its food, as in hypoglycemia, causes very serious brain damage. The patient with severe and prolonged episodes of hypoglycemia is likely to become feeble-minded or spastic, or both. We have seen that demonstrated so clearly in cases of diabetes in which the patients have been repeatedly overtreated with insulin that we feel urged to take up a little missionary work or education in the recognition of spontaneous hypoglycemia.

The symptoms and signs that occur in mild cases of spontaneous hypoglycemia are often so indefinite, so much like symptoms in other disease states, that it may be erroneously confused with the latter. Among children, the most frequent type of spontaneous hypoglycemia is primarily a disease of infancy, though occasionally it may persist into early childhood. We do not know as yet whether the idiopathic hypoglycemia among adults is of a similar nature. I am now looking for adult cases for parallel studies.

Figure 1 is a schema symbolically representing anatomic and physiologic factors which are known to regulate blood sugar levels. If you examine it, you will see it is not entirely whimsical, but summarizes our present knowl-

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Presented before the thirty-ninth annual Assembly of the Interstate Postgraduate Medical Association at Minneapolis.

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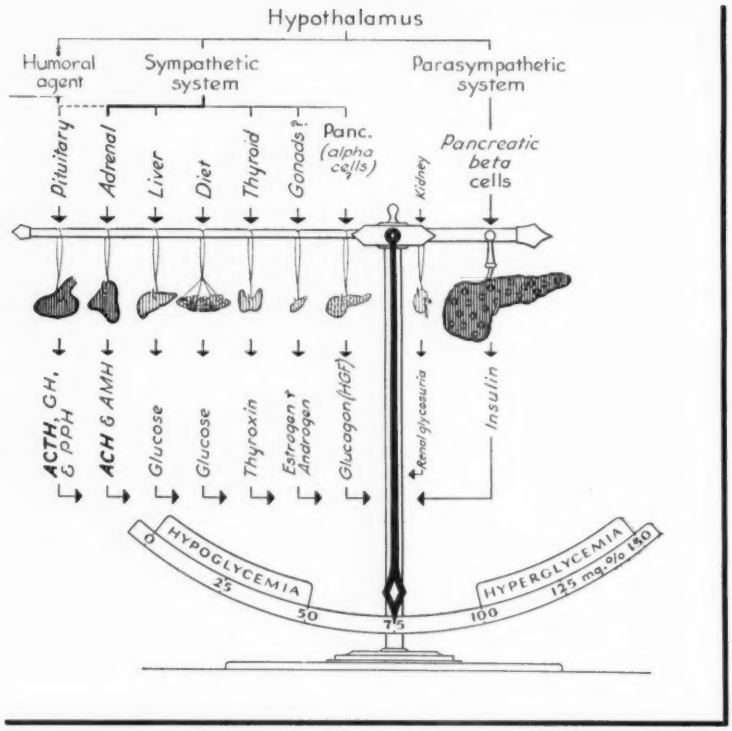

FIGURE 1. Symbolic representation of balance between hypoglycemic and hyperglycemic factors affecting carbohydrate metabolism.

From McQUARRIE, IRVINE: Idiopathic spontaneously occurring hypoglycemia in infants. A. M. A. Am. J. Dis. Child. 87:491, 1954.

edge very well. We have arranged here the many factors that can affect carbohydrate metabolism. Disturbance of these functions in one direction or the other may produce either hypoglycemia or hyperglycemia. At the top of the diagram is shown the hypothalamus with its special nuclei, which give rise to the two components of the autonomic nervous system. The beta cells of the pancreas which secrete insulin presumably receive their chief innervation from the parasympathetic system. The adrenals and probably some of the other organs which are shown to antagonize or balance the action of insulin are innervated by the sympathetic fibers. The anterior pituitary has a humoral rather than a nervous control by the hypothalamus.

The relationships are shown symbolically by use of an old-fashioned steelyard, modernized by attaching an indicator, and at the bottom, a scale, indicating blood glucose values (Smogyi method). Note that the lower limit of "normal" is considered to be 50 mg. per cent and the upper limit, 100 mg. Then we go into the range of hyperglycemia above 100, and that of hypoglycemia below 50 mg., the range which interests us most today. One could find explanations for most of the known causes of hypoglycemia (or of hyperglycemia, for that matter) by referring to this illustrative diagram.

In cases of tumor of the islands of Langerhans, or in beta cell hyperplasia without a distinct tumor, we have a definite explanation of hypoglycemia due to *absolute hyperinsulinism*. If it is a solitary benign tumor or several small discrete tumors, their surgical removal usually results in permanent cure. Malignant tumors with metastases obviously present much more difficult surgical problems. The hyperinsulinism due to simple hyperplasia of the pancreatic islands is relieved by partial pancreatectomy, at least temporarily.

You can see that if any of the hypergly-

cemic factors represented on the left in this symbolic balance are underactive, while the beta cells of the pancreas maintain their normal activity, there is hypoglycemia due to a *relative hyperinsulinism*. When either the pituitary body or the adrenal gland, each of which produces several hyperglycemic hormones, and the thyroid (possibly the pancreatic inner secretion, glucagon, as well) are overactive, while the pancreas secretes insulin at a normal rate, hypoglycemia develops.

Whatever the nature of the causative mechanism (endocrine, nutritional, neural, visceral or enzymatic), the symptoms and signs and the injurious effects of severe and prolonged hypoglycemia are the same. The first desiderata are its early recognition and prompt treatment before irreversible brain damage occurs.

Symptomatology and Diagnosis

In dealing with patients suffering from certain common clinical disorders known to produce spontaneous hypoglycemia, such as Addison's disease, pituitary insufficiency, severe hypothyroidism, von Gierke's glycogen storage disease and other forms of severe liver disease, the physician should be aware of the proneness to hypoglycemia, particularly after prolonged periods of fasting. The physical stigmata which characterize the majority of such patients aid in the diagnosis. An ameliorating response to feedings should suffice to suggest the presence of hypoglycemia in many instances, but determination of the blood sugar level at the proper time is essential for definitive diagnosis.

Other patients, particularly infants, without physical stigmata of primary disease, who present signs and symptoms compatible with, though not pathognomonic of, spontaneous hypoglycemia often present puzzling diagnostic problems to the physician. Because clinical manifestations of hypoglycemia are so vague and so irregular and fleeting in character, they are likely to be attributed to other functional disturbances, such as physiologic hunger, fright, "teething," excessive activity and loss of sleep. When the affected individual is fed with some carbohydrate food or drink, he is revived so completely for a variable period of time that members of the family at first feel certain that there is nothing wrong with the baby.

If symptoms or signs such as "wilting spells," irritability, nervousness, muscular twitchings, tremors and jerking, transient strabismus, rolling or glassy expression of the eyes, pallor of skin and mucous membranes, flushing, quickening of the pulse, cold clammy sweats and other vasomotor manifestations are seen in an infant or complained of by older children, hypoglycemia should be strongly suspected. If these indications are known to be ameliorated by meals or a glucose solution but not by water sweetened with saccharin, the occurrence of hypoglycemia is practically certain. The final proof is demonstration of low blood sugar values.

Onset of severe hypoglycemic episodes may first be manifested by a generalized convulsion or an attack of coma, though these phenomena are usually preceded by the milder signs. When the latter have not been observed by the family doctor and signs of acute illness are not present, he is prone to discount the significance of the first convulsion or to attribute the reaction to what the child has eaten (instead of what it has not eaten). When the distraught mother calls him again because of similar convulsive episodes, he is likely to misdiagnose the condition as epilepsy, for which he prescribes antiepileptic drugs, but usually without a favorable response.

When hypoglycemia is discovered, a battery of special tests may be made to determine more definitely the basic etiology of the disorder. These include (1) the epinephrine injection test for the presence of glycogen stores in the liver and for the intactness of the glycogenolytic function (special test for von Gierke's glycogen storage disease), (2) corticotropin (ACTH) test for glycemic and eosinopenic responses to test intactness of adrenocortical function, (3) epinephrine-infusion eosinopenic test for responsiveness of hypothalamic-pituitary-adrenocortical system to this stress agent, (4) glucose tolerance test and (5) insulin tolerance test.

When all the special tests fail to reveal any

positive result other than hypoglycemia with increased glucose tolerance and decreased insulin tolerance, the diagnosis of *infantile idiopathic hypoglycemosis* is warranted.

The principal features which characterize this clinical entity are as follows: (1) early age of onset (24 of 30 cases in our series began before the second year of life; only one began after the fifth year—hence the use of the term "infantile"); (2) absence of any peculiar physical stigmata; (3) all special tests for liver, adrenocortical, thyroid and pituitary insufficiency negative; (4) familial tendency (13 patients occurring in four unrelated families in this series); (5) natural tendency to spontaneous recovery or amelioration of symptoms with increasing age; (6) uniformly favorable therapeutic response to corticotropin (ACTH).

Treatment of Infantile Idiopathic Hypoglycemosis

I have three little patients here, in two of whom the condition was discovered very recently. The third is one with whom we have worked for some time. The two younger patients came to my attention recently through the courtesy of their physicians. These three little patients are all boys, and their ages are six months, 19 months and approximately two years.

This particular entity, being a disease of infancy primarily, is not likely to be seen by the internist, if the latter is living up to his usual practice of accepting adults only. The family practitioner is likely to get these cases first; the pediatrician may see them later.

We will just ask the mothers concerning the symptomatology. First, Mrs. J., will you tell the doctors here what you noticed first in your baby?

MRS. J.: Well, he had a staring condition and didn't seem to be breathing normally, and I would come in and find him in the crib staring straight ahead, and then he would be terribly limp. I thought at first he was just terribly relaxed, but his arms would immediately drop, he would have no tension in them at all, and ever since birth it seems he has been quite relaxed. The alarming situation was when he would stare and fix his eyes without moving or focusing them.

DR. MCQUARRIE: Did you see the doctor at that time, or did you postpone it?

MRS. J.: At the time of the baby's formal check-up, I had told the doctor about the baby's episodes of limpness and the staring, but we weren't alarmed about it at that time.

DR. MCQUARRIE: Then later the baby had convulsions?

MRS. J.: That's right. It developed into a muscular twitching at home and I immediately called the doctor. He had me bring the baby in, and from there we rushed him to the specialist who was to get the electro-encephalogram, which was to take a week. In the meantime he was put on a medicine to slow down the convulsions, but they continued, so I called the doctor again. He said I had best bring him to the hospital for observation, and that is when the low blood sugar was discovered.

DR. MCQUARRIE: Then he went on ACTH treatment?

MRS. J.: Yes, he was put on ACTH right away.

DR. MCQUARRIE (to audience): Remember those symptoms — staring, "wilting," some people say. Mrs. B. is next. Will you tell us what you noticed?

MRS. B.: Ever since Tommy was a baby, he didn't seem to develop properly; he wasn't strong.

DR. MCQUARRIE: He was a premature baby, the smaller of nonidentical twins.

MRS. B.: He would play a little while and then would get tired and act as if something were wrong with his heart, and finally one morning I went in and he was whimpering and I found he had this staring condition. He would pull his little arms up and have a sort of convulsion. We took him to the doctor and he thought maybe it was from teething with a mild spasm of some kind. This happened about three or four times and finally, the last time, it was found he had low blood sugar.

DR. MCQUARRIE: Then the baby had convulsions and coma?

MRS. B.: Yes.

DR. MCQUARRIE: Now let's hear from Mrs.

W. Those other two infants began to have symptoms with certainty at four months of age. When did your child have his first symptoms?

MRS. W.: Johnny was just about four months of age. He had the same glassy stare, but his eyes would cross and he looked as though he were actually in another world; he just didn't seem to focus on anyone or anything around him, and up to that point he had been noticing people and noticing everything. Of course, we brought him right to the hospital and found it was low blood sugar. He did have jerking of his arms and he had a few mild convulsions.

DR. MCQUARRIE: They recognized this early. As the family doctor, who is here today, knows, there were other previously discovered cases among Johnny's cousins. As a matter of fact, one older child, a sister, had a milder, more transient form of the condition at two years of age, but not severe enough to produce more than a few convulsions. By dietary treatment, watching her carefully over many months, she never required either operation or any very special medical treatment. She appears to be entirely over this hypoglycemic tendency.

This little boy is an active, essentially normal two year old now. He has been maintained on corticotropin (ACTHAR GEL®) since the first week of his manifestations of spontaneous hypoglycemia. There is one additional point I would like to make. This first little patient has a mild but transient skin eruption, an acneform lesion over the face. We regard this and a "cushingoid pudginess" as warnings of overdosage of corticotropin. They are nearly all rather fat babies due mainly to excessive eating and in some instances to excessive use of corticotropin, an unnecessary complication to which these young infants appear to be especially prone.

We have studied about 14 other little patients with the same disorder recently, in most of whom the mistaken diagnosis of epilepsy, birth injury, subdural hematoma or encephalitis had been made, without any definitive supporting history for those conditions. Large amounts of drugs were given until the patients were dopey and quite under the influence of the drugs themselves, but without control of the convulsions. We have several other cases of hypoglycemia which I shall describe later if time permits, cases of patients who have gone 6 to 12 months on conventional sedative drugs without any beneficial treatment. Now they have some permanent residuals of brain damage such as spastic states, secondary epilepsy and severe mental retardation, tragedies which can now be prevented by proper use of corticotropic hormone.

Of historical interest is the case of a boy who was referred to us in 1940 as an epileptic. Of course, "the woods are full of epileptics," a "snap" diagnosis which is made indiscriminately and too frequently for all sorts of convulsive disorders in children. We soon observed, however, that all his convulsions occurred in the morning. We made fasting blood sugar tests, as we do routinely in all convulsive cases, and found that his fasting blood glucose was 20 mg. per cent.

He was a pituitary dwarf with the somatic development of a normal child six years of age, although he was chronologically 12 years old at that time. We gave him some extra meals each day, but these did not abolish his fasting hypoglycemia entirely. He obviously had hypopituitarism, a state which made him tremendously sensitive to insulin, so that a normal or nearly normal insulin alone was sufficient to produce hypoglycemia with convulsions when he was in the fasting state (relative hyperinsulinism). He is especially interesting from the point of view of our subsequent successful use of ACTH therapy.

We fed him a lot of commercial pituitary gland products by mouth without detectable effects. Our reason for resorting to the use of pituitary extracts in the first instance was that Young and co-workers several years previously had induced permanent diabetes mellitus in experimental animals by prolonged intramuscular injections of crude anterior lobe extracts. We conceived that such diabetogenic material might be ideal for treatment of the opposite state, spontaneous hypoglycemia.

When we gave this boy a partly purified concoction of active principles from the anterior pituitary gland, called Polyansyn (Armour

and Company, no longer marketed), which could be given intramuscularly, he stopped having convulsions after a few days and his fasting blood sugar became normal. The injections of this product were given every other day for several weeks. He has never had any convulsions or other hypoglycemic symptoms since that time. That was over 14 years ago. He has remained cured, so far as his hypoglycemia and convulsions are concerned.

This amazing response was long thought of as a possible coincidence (a result "too good to be true"), but it set us to looking for hyperglycemic substances which we might use directly in cases of various types of hypoglycemia, long before either ACTH or cortisone was isolated.

In the case study of a second boy, who is now 11½ years old, we witnessed a great tragedy in that his convulsive state was not diagnosed as being caused by hypoglycemia until extensive brain damage had been done. He was given, in the meantime, an immense amount of antiepileptic drug by his doctors over a period of many months, but despite this he continued to have convulsions. A pediatrician in St. Paul then made the diagnosis of severe hypoglycemia.

Immediately they withdrew the drugs and had about three-fourths of his apparently normal pancreas removed, because the surgeon could find no signs of hyperplasia or tumor. He was then thought to be "cured" by this operation for several months, but again all his symptoms returned. Then a second operation was performed in which another portion of his pancreas was removed. That was the best treatment we had at the time. He remained symptom-free thereafter up to about four years of age, when he had recurrence of an occasional convulsion and other symptoms of spontaneous hypoglycemia.

The family asked me if it was inadvisable to have a second child—if this abnormality is ever hereditary. I assured them that to the best of my knowledge it was not hereditary. They had another baby, a lovely baby girl, whom I saw at six months of age. Like these other babies whom you have just seen, she appeared to be entirely normal before she had any manifestations of hypoglycemia. However, about six weeks later the mother called me and said very courageously that she knew from experience with her son that the new baby had hypoglycemia; she made the correct diagnosis herself. I asked her to bring the infant to my laboratory, which she did. The fasting blood glucose was found to be 18 mg. per cent. We used all the therapeutic resources in the book; we even used adrenal extracts, hoping to get the hyperglycemic effect from the adrenal cortex, but we did not give enough to do much good, and this baby had one convulsion after another. The blood sugar was as low at times as 8 or 10 mg. per cent, even while she was receiving meals which were reasonably high in carbohydrate, namely, a modified milk formula every three or four hours.

While I was away from home, the baby's hypoglycemia became so bad that the staff recommended, and the parents were pleased to have, a partial pancreatectomy, 75 per cent of the organ being removed. That resulted in complete freedom from hypoglycemic symptoms for about five weeks. Then, severe hypoglycemia recurred.

At this juncture, corticotropin (ACTH), which we had long desired to obtain for a special clinical test on hypoglycemic patients, became available to us for trial. When placed on this hormone, the patient and her still hypoglycemic older brother both responded strikingly to it. The eosinophil count was greatly reduced and the fasting blood sugar level rose to normal when ACTH was started. When it was omitted, the eosinophil count returned to the original normal level, whereas the blood glucose declined more slowly. This next point is important. Before treatment with ACTH was begun, the fasting blood sugar varied between 22 and 8 mg. per cent. The hormone was withdrawn after four days. Withdrawal of corticotropin (ACTH) was not followed by a drop to the low initial values; they stayed up for 8 or 10 days at intermediate or borderline levels above the reaction level, with the patients seemingly very much improved for the while. However, shortly after this period the values went down to hypoglycemic reaction levels again.

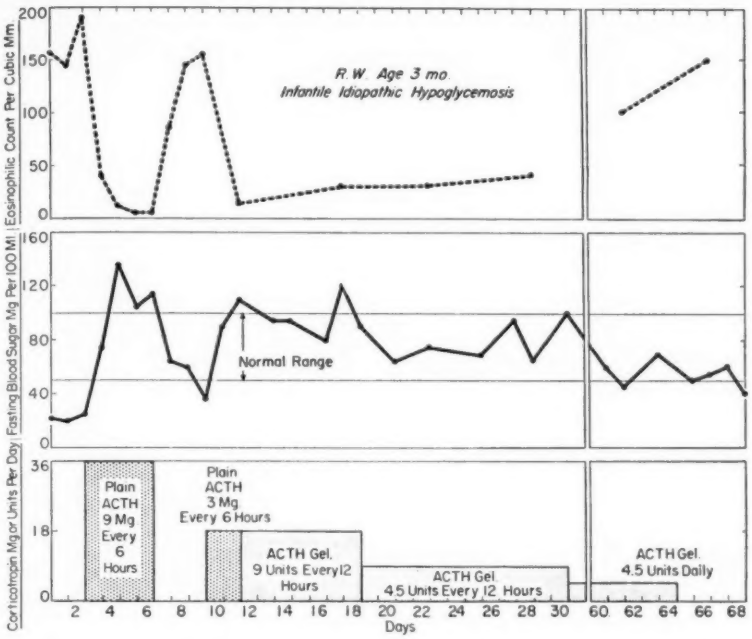
R.W. Age 3 mo.
Infantile Idiopathic Hypoglycemosis
Eosinophilic Count Per Cubic Mm.
Fasting Blood Sugar Mg. Per 100 Ml.
Normal Range
Corticotropin Mg. or Units Per Day
Plain ACTH 9 Mg. Every 6 Hours
Plain ACTH 3 Mg. Every 6 Hours
ACTH Gel. 9 Units Every 12 Hours
ACTH Gel. 4.5 Units Every 12 Hours
ACTH Gel. 4.5 Units Daily
Days

FIGURE 2. Effects of therapy with corticotropin on fasting blood glucose and eosinophil counts of a four and a half month old infant with idiopathic hypoglycemosis (hypoglycemic state). The child's history was uneventful up to the age of three months. Then, symptoms of low blood sugar with repeated convulsions appeared. Antiepileptic drugs were ineffectual for controlling convulsions. Decreasing doses of corticotropin were given over a period of 64 days. H. P. Acthar gel, in maintenance doses of 2 units once daily, was administered for two additional months. Therapy was then discontinued entirely without return of symptoms.

From McQUARRIE, IRVINE, ULSTROM, ROBERT A. and ZIEGLER, MILDRED R.: Random notes concerning the etiological mechanisms and treatment of spontaneous hypoglycemia. Acta paediat. 43:481-494, 1954.

We took advantage of this observation, which was experienced in four other cases as well. We decided that something had happened as a result of the intensive four day period of therapy that might be effective if we used smaller doses regularly in the post-ACTH period. So, we gave 18 mg. every 48 hours and maintained the fasting levels well above reaction levels. This attempt was successful in the first patient and subsequently with many others. With the gradual lengthening of intervals and reduction of dosage, this child, at the end of a year's treatment, was found to require no more corticotropin. That was four years ago and she has never had any hypoglycemia or symptoms of reaction since. She is normal in every respect. Her older brother is markedly retarded in every respect. He is somewhat spastic, does not hear or talk, and has had an occasional convulsion, even when the blood sugar was normal. We believe that these neurologic residuals of long-neglected hypoglycemia are attributable to brain damage, which can now be avoided.

This clinic can be concluded by illustrating the clinical course of a typical infant with idiopathic spontaneous hypoglycemosis under therapy with corticotropin (figure 2).

Esophageal Hiatal Hernia

DONALD B. EFFLER*

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DONALD B. EFFLER

My approach to the subject of esophageal hiatal hernia has a very deep missionary zeal. I say that not because I think I have the answer to the problem or because I think that I handle the surgical aspects of the disease better than anyone else; the reason is that we are, by default, letting the management of esophageal hiatal hernia drift away from the field of medicine and into the field of the patent medicine vendor.

A great many people are using various patent antacids because they have heartburn, they are dyspeptic, and they are tired of going to doctors and paying fair amounts of money and not getting any explanation and, worse yet, not getting any treatment.

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Presented before the thirty-ninth annual Assembly of the Interstate Postgraduate Medical Association at Minneapolis.

Diaphragmatic hernia, particularly esophageal hiatal hernia, is a common lesion. Not all hiatal hernias produce symptoms, but the point I wish to make is that they cause more trouble than we have been led to believe. The only difference between them and other hernias of the abdominal cavity is that they do not bulge out under the belt; you cannot see them and you do not have to wear a truss. But the radiologist can see a hiatal hernia if he looks for it. We can suspect the lesion if the patient is having symptoms when he comes in the office, and I think we ought to recognize that it is potentially capable of costing the patient his life.

Over the years, the subject of hiatal hernia has been included in every textbook on gastroenterology, and it is the gastroenterologist at whom we must point the accusing finger. It is he who should have started the ball rolling but whose attitude has led to a certain apathy in the medical teachings on the subject. He could not see the disease very well; it was an x-ray curiosity primarily, and none of the medical treatments worked. The patients were ungrateful after following a long

program of medical treatment, so the gastroenterologist shoved the condition into obscurity and said, "If the patient would get his mind off it, there wouldn't be much to worry about."

I shall outline briefly some of our own observations and experience. I have seen some 500 patients with hiatal hernia in the past eight years, and we have operated on more than 200 of them. Our findings have been very interesting, and I would like to pass some of them on.

First let us consider symptoms and differential diagnosis. There are two symptoms which are present in every case of *symptomatic* hiatal hernia (and not all of them produce symptoms): (1) pain, heartburn or retrosternal discomfort after eating, and (2) pain on recumbency. The former can easily be distinguished from the pain of duodenal ulcer, as the latter produces discomfort three hours after eating. Also, the patient with hiatal hernia feels better when his stomach is empty, whereas the patient with duodenal ulcer feels worse. Both get relief by taking antacid. The pain on recumbency also is relieved by antacid; the person with symptomatic hiatal hernia awakens at night flat on his back and has heartburn, gets up, walks to the bathroom, and takes a glass of water and a patent alkalizer (they seem to prefer these to the medical preparations).

In many textbooks and medical presentations, symptoms are confused with complications. This is important, because when we get into the complications we are getting into serious problems. There are many complications in hiatal hernia, but the three major ones are bleeding, ulceration and obstruction. Bleeding is the most common, and can occur in many forms—melena, hematemesis of tremendous amounts of blood. The hemoglobin can go down to less than a third of normal, and the patients can exsanguinate. Perhaps the commonest form of bleeding in hiatal hernia is that in which a very small amount is lost each day and the patient is not aware of blood loss.

Peptic ulcers of all types can develop within the thoracic stomach, and these can perforate and they can hemorrhage, and the patient with this complication needs immediate treatment.

In the third type of complication, some mechanical obstruction occurs—usually volvulus. The hernia can get so twisted and incarcerated that it shuts off its own blood supply and causes periodic vomiting, stricture formation, and so on.

Before going any further, I would like to clarify one point. I think that one of the biggest mistakes made in this field of medical practice has been the attempt to correlate the symptoms and importance of the lesion with its size. For some reason, all our friends in gastroenterology (and I have very few of them left) will say that if the hernia gets large enough by all means take care of it, but if it is small the patient can prop her bed up and sleep in an upright position (if her husband will allow her), and, "If they take the particular management I am going to outline around the clock, 24 hours a day, there is no reason for doing surgery and curing them."

It is impossible to correlate the size of a hiatal hernia with its clinical significance. Some patients with tremendous hernias are perfectly happy; they just belch a bit more than other people. On the other hand, some patients with tiny little hernias are nearly exsanguinated from them.

To be an expert at anything, one must have a classification. I do have a classification, and it is getting simpler all the time. Figure 1 is a diagram which illustrates the three major types of hiatal hernia—para-esophageal hiatal hernia, pulsion type hiatal hernia, and the thoracic stomach associated with a short esophagus.

In the para-esophageal hernia, the esophagus is normal in length, and the stomach herniates up through the hiatus. The pulsion type is basically the same as the para-esophageal hernia, except that in the former the esophagus is telescoped by the herniating stomach. Clinically the two behave the same, but to be precise there is a slight difference in their radiographic impressions.

The thoracic stomach associated with a short esophagus presents the most serious problem. The distance down through the

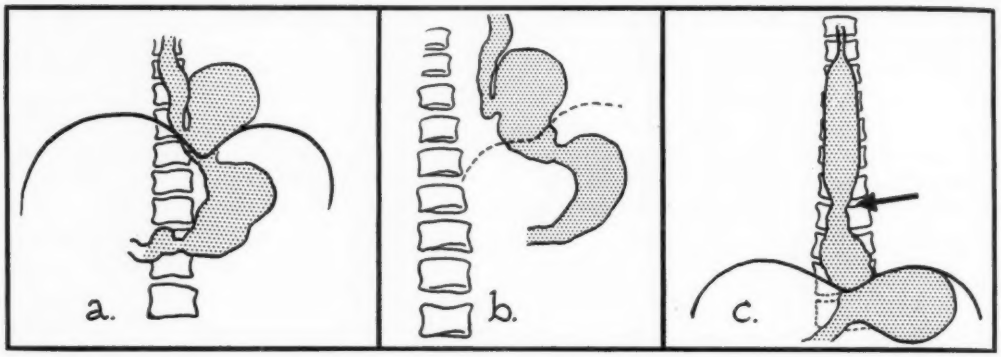

FIGURE 1. There are three major types of hiatal hernia, and the above diagram is intended to demonstrate the various types as they appear on routine barium contrast films.

a. The para-esophageal hiatal hernia in which the esophagus is of normal length and the herniation of the stomach occurs in front of the esophagus.

b. The pulsion type hiatal hernia in which the esophagus is "telescoped" by the herniated stomach. Actually the esophagus is normal in length, but is simply displaced vertically by the thoracic stomach.

c. The thoracic stomach associated with short esophagus. The arrow indicates the actual location of the cardiac end of the esophagus. In such a situation the esophagus is actually shortened, usually by muscular contraction and inflammatory changes associated with periesophagitis. The degree of shortening varies with the extent and severity of the inflammatory process. This lesion is frequently referred to as "congenital short esophagus." There is evidence now to suggest that the majority are acquired rather than congenital.

thorax to the diaphragm is too great for the short esophagus. As the shortening process progresses, it literally drags the stomach up along with it. In the pulsion type, the intra-abdominal pressure pushes the stomach up. For some reason unknown to me, this is called a congenital short esophagus, and nothing could be further from the truth. Congenital short esophagus is actually quite a rarity. The short esophagus seen in this type of hiatal hernia usually develops in persons more than 35 or 40 years of age, and we believe, with good reason, that they are acquired lesions. They present quite a problem from the standpoint of therapy, and I shall discuss that later.

Many hernias do not fit into this classification, and one can give them various names, although I hesitate to use terms here that have little meaning and serve only to confuse the literature further. There is one type that I call the "balloon" hiatal hernia, simply because it is so large that a plain x-ray discloses it. You do not need a gastrointestinal series or an expert radiologist to diagnose such a hiatal hernia; all you do is listen to the patient belch once or twice or use the fluoroscope and the diagnosis is made. The tremendous tampon of air displaces its equivalent amount of alveolar air in the lung by crowding in the mediastinum, and also pushes the cardiac silhouette over anteriorly. It is easy to see why such patients progressively become more and more short of breath.

Another type probably could be called a "super-balloon" or "compound" hiatal hernia, because more than just the stomach comes up above the diaphragm. In one case, that of a young woman, roentgenograms made after a barium enema disclosed that in addition to stomach there were loops of colon going up through the hiatus, making a complete swing and coming back down again.

The predominant feature of another case was moderately severe volvulus in a large hiatal hernia. The stomach was looped upon itself in such a way that there was mechanical obstruction at all times. The patient for many years had regurgitation and vomiting after every meal.

In another case the volvulus was even more

severe. All the stomach was displaced above the diaphragm; the pyloric ending of the stomach was situated just below it. The patient had multiple fluid levels, and taking some medication and propping her bed up and a few other routine procedures would do very little to relieve her symptoms.

If the two cases just mentioned were severe types of volvulus, the next one could be called horrendous. This patient had an upside-down, loop-the-loop hiatal hernia. The cardiac end of her stomach was below the diaphragm, and it was the pyloric antrum which herniated through the hiatus. This condition was a potential death trap; a closed-loop type of obstruction could occur at any time. This patient could not vomit because of obstruction of the esophagus, nor could she empty the stomach because of obstruction of the pylorus. If the stomach became ischemic, spontaneous rupture would occur.

Patients such as the one just mentioned represent emergency cases, and loss of life is imminent. Even if one is aware that a patient has a hiatal hernia, one must remember that often the diagnosis is extremely difficult because the patient's condition is catastrophic when he is seen. More than any other condition, the situation resembles acute pancreatitis. I almost put that label on the last-mentioned patient when I first saw her. Surgical management in such cases, of course, frequently includes resection.

I shall describe briefly some complications encountered in cases of hiatal hernia. Obstruction occurred in one case due to an ulcer right at the hiatus. An x-ray taken when the patient was admitted for emergency treatment showed the thoracic stomach full of barium. A film made four hours later disclosed that the barium was bouncing back and forth. Constriction was noted due to edema of the stomach wall from the ulcer, and the barium was slowly trickling down into the stomach. This case constituted a very real emergency, and such patients require prompt surgical intervention.

Another very real complication is represented by the case of a patient whose colon actually entered the esophageal hiatus. A horseshoe-shaped lesion visualized on x-ray actually was a normal transverse colon, but it was in the mediastinum behind the cardiac silhouette. The result was that this patient had a tremendous cardiac silhouette with actually quite a small heart. His stomach, colon, spleen and small bowel all entered the hiatus. Significantly, he had very advanced emphysema, which I am sure was mechanical and due to displacement of the major mediastinal vessels. Even though such a condition could be surgically repaired, in this instance the many years of mechanical pressure resulted in cor pulmonale, and the patient did not survive more than six months after operation.

Now we come to complications of the type of hernia associated with a short esophagus. We have seen about a dozen cases of this type in which gastric ulcers developed. In three, perforation occurred; one perforated into the mediastinum, and the other two had perforated slowly into the lung, resulting in an unusual type of fistula. These patients are critically ill when you encounter them.

A very severely complicated type of short esophagus was seen in a patient with mechanical fibrous stricture and a large gastric ulcer. In addition, he had very severe bleeding, so all three of the major complications of hiatal hernia were present. These patients are literally gastrointestinal cripples. The only way to treat them when they reach this stage is as though they had a malignancy—doing a very radical resection and esophagogastrostomy. That is the only type, however, except for acute perforating ulcer, that requires resection.

The ulcers that we have seen in cases of hiatal hernia are usually deep craterlike ulcers and usually occur in the gastric mucosa immediately below the terminal end of the esophagus. The mucosa here usually is thickened or there is tremendous hypertrophic gastritis that bleeds easily.

I will outline briefly my concept of the mechanism of the pain experienced by patients with hiatal hernia. The duodenum, of course, is alkaline; the antrum is slightly on the alkaline side, and mucin is also secreted. The rest of the stomach secretes acid. When a person with a normal stomach eats, he has no pain afterward as long as his stomach is not

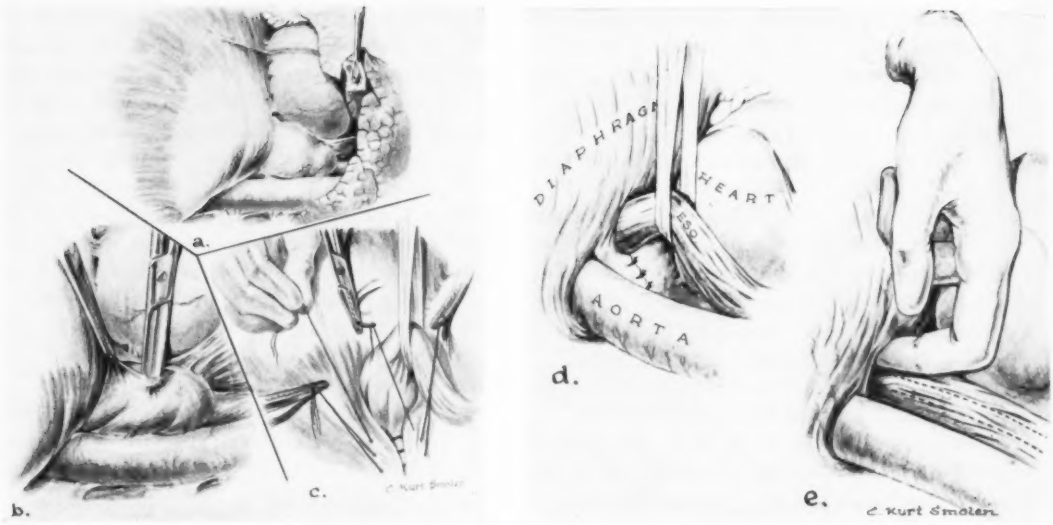

FIGURE 2. Diagram illustrating technic of surgical repair of para-esophageal or pulsion type hiatal hernia. A transthoracic approach is employed under general anesthesia. The surgical correction follows the simple requisites of all hernia repair regardless of site.

a. Demonstrates the relations between the hernial sac and the adjacent structures including the diaphragm, pericardium, left lung and aorta.

b. The mediastinal pleura is opened and by sharp and blunt dissection the thoracic stomach and terminal esophagus are completely mobilized.

c, d and e. Demonstrate the approximation of the right and left crura of the diaphragm behind the esophagus to close up the esophageal hiatus. The Levin tube is left in the stomach for suction.

Usually a small counterincision is made in the dome of the diaphragm and the cardiac stomach is fixed to the inferior surface of the diaphragm for added security. This is not shown in this series of drawings.

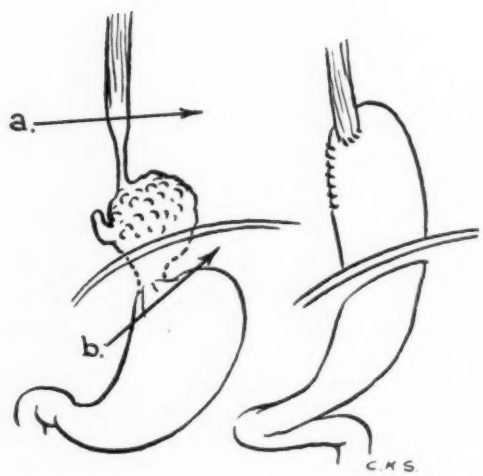

FIGURE 3. In those patients with severely complicated short esophagus, extensive peptic ulceration may destroy the integrity of the terminal esophagus and cardiac portion of the stomach. When irreversible changes occur, primary resection and anastomosis will be necessary.

The basic type of therapy is illustrated here. The lines a and b represent the lines of resection, and the anastomosis between esophagus and remnant of stomach is shown on the right.

This form of therapy is by no means the desirable form of treatment, but will be required in a small percentage of cases in which local disease has progressed beyond point of recovery.

(Figure 2 is reproduced courtesy of the *Journal of Thoracic Surgery*; figures 3, 4 and 5 courtesy of the *Journal of the American Medical Association*.)

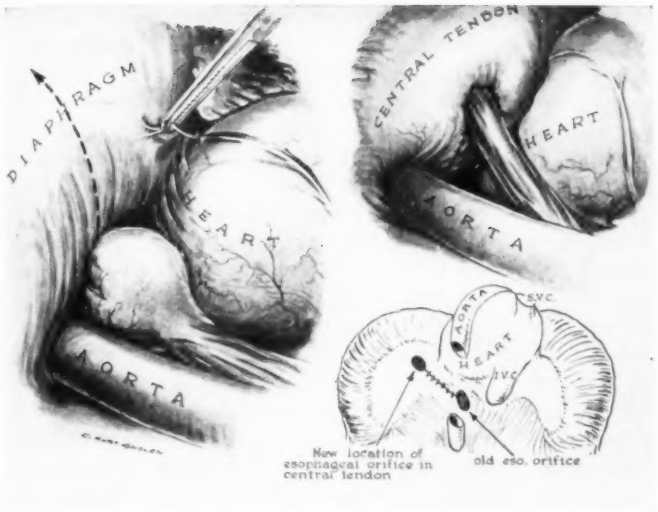

FIGURE 4. Surgical correction of short esophagus without resection can be accomplished in the manner outlined here. This involves permanent paralysis of the left leaf of the diaphragm and transplantation of the esophagus to the central tendinous dome of the diaphragmatic leaf. If proper dissection is performed, reduction of the thoracic stomach below the diaphragm in this manner can invariably be done. When the stomach is reduced below the diaphragm permanently, the symptoms associated with short esophagus are promptly relieved.

overdistended and the food goes into a liquid state. There is a uniform pH, you might say, that is well tolerated on the acid side, and there is a constant reflux regurgitation of alkaline juices and a constant buffering by the food, by the juices, by the mucin, and so forth, to prevent the normal stomach from digesting itself. This, then, is good digestive balance.

If a person has a short esophagus, however, or a hiatal hernia, the gastric mucosa which is dragged up by the esophagus or pushed up in the hernia is that which contains, by and large, the greatest amount of acid-bearing, acid-secreting mucosa. None of the alkaline material goes up with it. When such a person eats, all the food goes below the diaphragm, unless there is extreme volvulus; it goes into the liquid state and is well buffered. But above

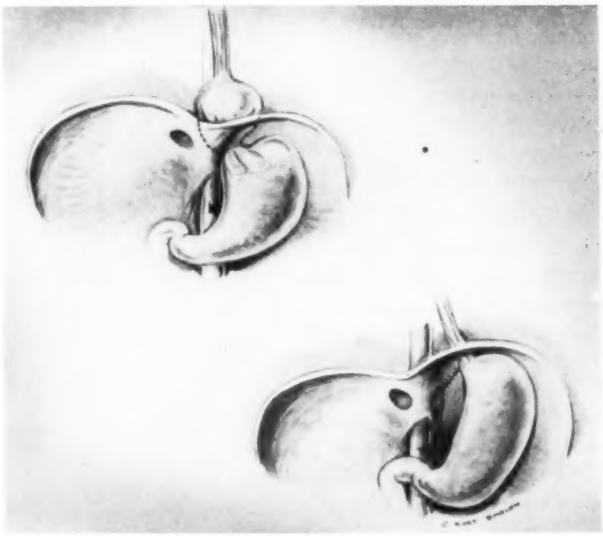

FIGURE 5. The principle of reduction of the thoracic stomach associated with short esophagus is demonstrated in this sketch. By elevation of the diaphragm and transplantation of the esophagus to a new hiatus, the previously shortened esophagus is now of adequate length and the entire stomach remains below the diaphragmatic level. This simple concept is the basic premise in the therapy of short esophagus.

the diaphragm there is literally a gastric pouch that contains no food, no buffering agent, but which, quite normally, in response to the digestive process, begins to elaborate acid which is not buffered locally and simply results in local digestion or local chemical reaction, if you wish, in the thoracic stomach. I believe that is the major mechanism for heartburn. This is attested to by the fact that this gastric mucosa is always found to be markedly inflamed, more friable. It bleeds very readily in contrast to normal mucosa. Also, if you perform the type of surgical procedure that we employ and put this portion of the stomach below the diaphragm so that the patient has one entire stomach instead of the hourglass type, prompt and lasting relief of the symptoms occurs.

The mechanism of heartburn on recumbency can be explained as follows: While a person with a short esophagus is in a recumbent position, his stomach is secreting, but it is buffering itself and there is no discomfort in the portion of the stomach below the diaphragm. But when one looks at an x-ray taken after a barium meal with the patient in a perfectly flat position, one sees the hourglass type of constriction, actually a definite pouching in the thoracic stomach. Again, the mucin that normally counterbalances or buffers the acid secretion is not secreted in this portion of the stomach. Also, the regurgitation from the duodenum, which is one of the important means of counterbuffering, does not occur in the thoracic stomach, and this person pools the secretions which are predominantly acid. In addition, there is a tendency toward a little esophageal reflux which accounts for it. If this person has pain in the recumbent position and stands to an upright position or simply takes a glass of water, this residue falls below and he obtains prompt relief.

The surgical management of hiatal hernia employed by us is illustrated in figures 2 through 5. We believe all surgical procedures performed for hiatal hernia should be done through the thorax. The reason for that is that you are going to encounter a certain percentage of cases in which there will be a very short esophagus, and if you are performing the operation from below you can do nothing for these patients. The transthoracic approach is, I think, pretty well established throughout the country. With good anesthesia, anyone who is trained in this type of surgery has very little difficulty as far as lasting repair is concerned.

Surgical correction of hiatal hernia embodies the same principles as any repair of hernia. When the dissection is complete, the thoracic stomach is reduced below the diaphragm, and the crura are approximated posteriorly to the stomach. When the operation is over, the stomach is below the diaphragm, the aorta and heart are not in any way interfered with, and the crura are approximated.

Surgical correction of short esophagus involves permanent paralysis of the left leaf of the diaphragm and transplantation of the esophagus to the central tendinous dome of the diaphragmatic leaf. You lengthen nothing; you remove nothing. All you are doing is changing relativity, actually. After operation, the hiatus which is normally low in the diaphragm is moved approximately 3 to 4 in. higher, and an esophagus that was too short is perfectly adequate. These patients obtain complete relief of symptoms attributable to the thoracic stomach.

DIAGNOSTIC CLINIC

Pancreatic Lesions

WALTMAN WALTERS* AND DIGBY CHAMBERLAIN**

Mayo Clinic, Rochester, Minnesota, and University of Leeds, Leeds, England

WALTMAN WALTERS

DR. WALTMAN WALTERS: It is my pleasure to be able to participate in a clinic with Professor Digby Chamberlain of Leeds, England. We are going to try to make this as interesting as possible, but due to the inclement weather the last day or two, and the difficulties in transporting patients, we have had to improvise a little. However, what we are going to say is really bona fide.

I am going to write down the different lesions of the pancreas on the board while Mr. Chamberlain begins the discussion.

MR. DIGBY CHAMBERLAIN: The first patient is the girl on the stretcher here who developed acute abdominal pain this morning. She tells us that she has had little attacks of upper abdominal pain before, but that no attack has been anything like the one that developed this morning. This pain was acute and upper abdominal, and she is just a little bit cyanosed. She also has a little rigidity in the upper abdomen, and the condition that we have in mind is acute pancreatitis. The symptoms are those that you would expect—a type of pain such as she has complained of, with a history of previous minor attacks. The acute attack is usually very severe and it is

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Presented before the thirty-ninth annual Assembly of the Interstate Postgraduate Medical Association at Minneapolis.

accompanied by vomiting. The vomiting is of some importance, because one must distinguish it from the vomiting that occurs in acute intestinal obstruction.

The differential diagnosis of this condition is one of the difficulties. In acute intestinal obstruction there will be two things which are not found in acute pancreatitis: (1) constipation or complete constipation, and (2) an increase in bowel sound, if you listen to the abdomen; that does not happen in pancreatitis. The cyanosis is almost certainly due to limitation of respiratory movement; there is no alteration in the blood which accounts for it.

When you try to come to an exact diagnosis, the points which will help you are the history, which I have given you; the question of vomiting, of which I have spoken; and the serum amylase test. The serum amylase is raised very considerably in the first 24 hours, after which it tends to revert to normal. The other tests are not as diagnostic as the serum amylase is.

One other point which is of help in the diagnosis is the straight x-ray of the abdomen. A straight x-ray of the abdomen is taken partly to exclude the presence of a perforated ulcer, because in the latter condition you would expect gas under the diaphragm, and, of course, this is not the case in acute pancreatitis.

One other finding in acute pancreatitis is a dilatation of a localized loop of small intestine in the upper abdomen, and it has been suggested that dilatation is due to the local action of pancreatic enzymes which escape and bathe that piece of small intestine, causing a paralytic distention of that localized portion.

The diagnosis, then, should be reached in those ways, but there is no doubt whatever that diagnosis is difficult and that many patients are operated on without the diagnosis being known. The facts are that the diagnosis in acute pancreatitis is arrived at by the general practitioner in my country in only about 10 per cent of the cases, and even the surgeon, with all the facilities of the hospital, arrives at an accurate preoperative diagnosis in only about 40 per cent of the cases. That, of course, must color the treatment.

There is no doubt that the treatment should be conservative. Conservative treatment carries considerably less mortality than operative treatment, and, if you operate, the less you do the better the chance for your patient. In fact, in cases in which the abdomen is opened and in which the surgeon has the courage to close it straightway, the mortality is about half of the figure it is if he does anything, whatever it may be. There is no justification for draining or removing the gallbladder, for draining the common duct, or anything else, and I am quite certain that the main reason for carrying out those operations is so that the surgeon can justify the fact that he has opened the abdomen.

As far as the treatment goes, conservatively it should be to rest the pancreas as completely as possible, and therefore nothing should be given by mouth. A stomach tube should be put down and kept down and aspirated. The patient should be on intravenous fluid, and if that fluid contains glucose it is important to give insulin, because the insulin activity of the pancreas is reduced when acute pancreatitis is present.

One other point which has been made in the prognosis and treatment of pancreatitis is the question of the serum calcium. The serum calcium has a normal reading of between 0.09 and 0.11, and in acute pancreatitis that level tends to drop. It has been said that if it drops to 0.07 per cent, the outlook is serious or almost invariably fatal. That probably indicates the severity of the disease—the fact that calcium is being used. This perhaps raises the question whether it might not be advisable to give calcium in addition to the other medication in the disease.

You know, of course, the possibilities of blocking the sympathetic. The reports of the results of that are varying, but that seems to limit the activity of the pancreas.

The main point I want to make is that treatment should be conservative. Both Dr. Walters and I have made up our minds that we are not going to operate on this girl.

DR. WALTERS: Now let us assume that this young woman is not too seriously ill but has

this attack of severe epigastric pain. She has had other attacks. Her history does not suggest a gastrointestinal lesion other than the disturbances of motility associated with disease of the gallbladder.

What do we gain by showing a patient here? In the first place you observe how the patient looks. She is young; she is not overweight. She does not look sick. In examining her abdomen, she does not have any particularly great tenderness although the right upper quadrant of the abdomen is tender to pressure. There is a possibility of biliary colic, but that is not too great because she has not had any medication that would incite the pain. The point I want to make in showing this young woman is that you should examine such patients carefully.

A few days ago I was in Nova Scotia and I was asked to see a nurse who had a history suggesting an upper abdominal inflammatory lesion. She had been ill for two or three weeks. As a visitor in that hospital I didn't want to do too much; I felt of her abdomen, and there wasn't any rigidity. Then I said, "Well, maybe we had better pull your nightie up and examine your abdomen." I had almost missed something that I should not have missed by trying to be a little bit modest. The patient had a very evident swelling in the epigastrium which was not discernible as I palpated her abdomen or as I looked at her abdomen through her nightie. As I looked at it, I could see a few slight peristaltic waves. I listened to her abdomen through a stethoscope and it was a real musical box. She had had two or three previous operations. I thought, this woman probably has a pyloric obstruction, she has had a difficult time, and she has been swallowing air. My friend in Nova Scotia, an astute clinician, and I talked it over and he said, "I don't think so."

What was the next thing to do? As Mr. Chamberlain said, a plain roentgenogram was taken of the patient's abdomen. There was no air in the stomach, there was no particular distention of loops of intestine, and the probabilities are the patient had a chronic inflammatory lesion of the pancreas with a cyst.

Now, Mr. Chamberlain, let's switch from acute pancreatitis to pancreatic cysts. Won't you please give us your views? We are trying briefly to cover the important points concerning lesions of the pancreas with the idea of helping in the diagnosis, if we can, and then to suggest what to do about it.

MR. CHAMBERLAIN: When we think of pancreatic cysts, two main ones should occupy our attention. The first, probably the commoner, is the pseudopancreatic cyst which follows recognized or unrecognized attacks of pancreatitis. This cyst usually gives a large swelling, filling up the epigastrium and is huge. The other type of cyst, the cystadenoma, is very much smaller. It occurs somewhere in the body of the pancreas and usually manifests itself by a certain amount of rather indefinite epigastric pain.

The diagnosis in the first one is usually fairly evident because it really resembles an ovarian cyst occurring in the upper part of the abdomen, whereas the cystadenoma is a small tumor which you may or may not be able to feel. In the cystadenoma the diagnosis is usually made by some distortion of the duodenum on radiology or possibly the finding of such a cyst when the abdomen is opened for unexplained, continuing discomfort.

The treatment of those two conditions varies. The pseudopancreatic cyst is one outside the pancreas filling up the lesser sac and its removal is usually impossible, or even if it would appear to be possible it is usually hazardous. We recommend that the cyst should be stitched to the abdominal wall and drained, the so-called marsupialization. Drainage in such a case may be prolonged, but it is, I think, the only method by which one is likely to get rid of such a thing.

It has been advised that anastomosis between the cyst wall and some part of the intestinal tract should be carried out so that one may get continuous intra-abdominal drainage instead of a fistula on the surface. Such operations, I think, are not safe, and my own advice would be against them. I think marsupialization must be the standby in such a condition.

In cystadenoma the condition is different. The cyst is usually small, and it is usually possible to dissect out such a cyst without

leaving a pancreatic fistula. Removal of part of the pancreas is usually not necessary. The lesion is a purely localized one, and therefore a careful dissection around the cyst is all that is necessary and should remove it adequately.

DR. WALTERS: Before you sit down, Mr. Chamberlain, I would like to discuss two cases. (We have not rehearsed these discussions.) I operated some time ago on one of these patients who had a large pancreatic cyst surrounded by a great deal of inflammatory reaction. I marsupialized the cyst. During the period of contracture of the cystic cavity a radiopaque, nonirritating substance was injected and a roentgenogram then showed that the main pancreatic duct emptied into this cystic cavity. We continued the drainage with a small catheter for months, but whenever the catheter was removed the fistula would close and the man would have pain and fever. What would you do in such a case?

MR. CHAMBERLAIN: I think that in such cases you will find that the opening into the pancreatic duct will eventually close, and the point that I make is that drainage in such a case should be prolonged. I know of a case of a doctor who used to go about with a tube in his cyst; he did his full work and he had the tube in his cyst for something like 18 months. The drainage can be prolonged, and my own feeling is that if one will be patient and continue the drainage, the likelihood is that the cyst will disappear. I do not think that any attempt at closing the duct is likely to be satisfactory, nor do I think that removing the cyst in its entirety is advisable, although I myself have done that operation for a pseudocyst on one occasion, with a perfectly satisfactory result.

DR. WALTERS: This is what happened in this man's case. The tube was kept in for about a year, and in the course of events when we removed it, the fistula closed and pain and fever recurred. Therefore I coned out the fistulous tract and sutured it into the stomach and everything was fine for another year. Then the patient had evidence of an acute upper abdominal inflammatory lesion. His physician said, "I think I feel a mass." He came back to the Mayo Clinic and we felt the mass. He had a cyst about a third the size of the original one, so I did a cystojejunostomy with an entero-anastomosis between the two loops of the jejunum so that food would not enter the cavity of the cyst. I had never done that before, as I hadn't encountered a case like this, but it seemed to me a rather interesting course of events.

I wish to speak about the second patient, because of the interesting features, and, as you know, we learn from our mistakes.

This patient, who is now in the hospital, is a young girl of 14 years who had been in an automobile accident in which two other occupants of the car were killed. She had several severe fractures (her jaw, some ribs and her pelvis). About a week or 10 days after the accident a mass was noted in the right side of the abdomen, and her doctor (who did a marvelous job of taking care of her in a small town in Iowa) made an incision in the right lower quadrant of the abdomen down to the cyst and marsupialized it. The girl improved, and the mass disappeared with the tube in place. Radiopaque material was injected into the fistulous tract by her home physician and the roentgenogram then seemed to show that a fistula terminated around the head of the pancreas. It seemed to me the probabilities were that the patient had a pancreatic cyst. When the tube was taken out or closed she would be sick. I decided that I would open up the cystic cavity. At operation disruption of the ureter from the pelvis of the kidney was found; the excretory fluid was urine. The cavity, drained, seemed to her home surgeon to be a pancreatic cyst. Yet it wasn't, the anterior part of the cyst being the posterior parietal peritoneum. We made our mistake by not examining the fluid which was being excreted. Moreover, the quantity of fluid drained was far in excess of the quantity that drains from a pancreatic fistula.

Two points come to mind: (1) Pancreatic fistulas usually do not present in the lower part of the abdomen; (2) before a diagnosis of pancreatic fistula is made, examine the secretion for enzymes. Be sure one way or another either that the drainage is pancreatic fluid or that it is not.

Wouldn't you like to make a comment or two on that case, Mr. Chamberlain?

MR. CHAMBERLAIN: The first case is interesting, and the fact that the fistula closed rather falls in with what I have said—that I am not, on the whole, in favor of internal drainage. In the second case, I quite agree with what Dr. Walters has said, that fistulas do not present in the lower abdomen; the swelling is in the upper abdomen, you open it in the upper abdomen, and it is in the upper abdomen that it drains. It is quite obvious that in any fistula of any kind you want to know what is coming out, and with a mass in the right side of the abdomen, the giving of one of the dyes excreted by the renal parenchyma would appear to be the method of solving the problem.

DR. WALTERS: Now we will take up the question of the treatment of chronic pancreatitis. Dr. Stanley Hoerr of Cleveland was kind enough to tell us of his discussion, and we regretted that we could not be here to hear him. In one of his slides, as I understand it, he enumerated the methods of treating chronic relapsing pancreatitis.* What are your methods, Mr. Chamberlain?

MR. CHAMBERLAIN: I think that is a surgical problem which is not solved. There is the variety of pancreatitis, of course, which mimics carcinoma, which presents a painless jaundice with enlargement of the gallbladder. There the differential diagnosis, again, is difficult. There is nothing in the history, in the findings at operation, or in the biopsy which will really help you, because when you do a biopsy of such a pancreas it is unusual to get the cancer cells. You tend to be just a little bit conservative, you do not want to cut into the body of the pancreas because of the danger of injuring the splenic vessels or the main duct, and you nibble out little bits, which are not characteristic of anything. It is quite true that you may be fortunate and get a piece that shows carcinoma and then the result is quite evident.

In the majority of these cases the diagnosis is made only after the lapse of time when you find that something has been done and your patient is still alive. That is not the main problem. The main problem is the patient with these recurrent attacks of upper abdominal pain in whom you may find areas of calcification within the pancreas. The procedures that have been done have varied from removal of part of the pancreas to the blocking of the sympathetic, but in my experience they are perhaps the most difficult cases that we have and I would like to throw the ball back to Dr. Walters.

DR. WALTERS: The diagnosis and treatment of chronic relapsing pancreatitis and its surgical handling constitute an important problem in abdominal surgery. Several procedures can be employed but in my own experience good results can be expected in only about 75 per cent of the cases from any method.

Theoretically, cutting the sphincter of Oddi transduodenally will relieve the intrabiliary pressure and hence reflux does not take place into the pancreatic duct. The theory behind anastomosis between the bile duct and the duodenum or the bile duct and the jejunum is the same, plus a consideration of the fact that in some cases the lower portion of the bile duct is narrowed. I have seen and demonstrated that in some cases of pancreatitis in which sphincterotomy has been done and a short T tube used to study the physiology of the common duct afterward, the lower portion of the common bile duct is still narrowed because edematous pancreas snuggles around it. Choledochoduodenostomy has been effective in about 75 per cent of these cases.

Two years ago in a Mayo Foundation Lecture, Mr. Kenneth W. Starr of Australia described results obtained with sphincterotomy with localization of the major pancreatic duct at its entrance into the ampulla and catheterization of this duct. In many of those cases he found narrowing of the pancreatic duct just beyond its entrance into the ampulla, and he dilated that. This together with the sphincterotomy gave excellent results in approximately 75 per cent of the cases.

In Nova Scotia last week I saw such a patient. An astute surgeon in Boston had palpated

*HOERR, STANLEY O.: Diseases of the pancreas. Postgrad. Med. 18:32 (July) 1955.

a distended main pancreatic duct, catheterized it after dividing the sphincter of Oddi, put a little catheter in the pancreatic duct, injected a radiopaque substance later, and demonstrated the dilatation of the duct. The patient has been relieved of his symptoms. It is not too long a time after the operation, but the whole procedure indicates thoughtful analysis of the conditions that existed.

I have spoken about internal biliary drainage by choledochoduodenostomy. Then there is external biliary drainage by means of a T tube inserted into the common duct. If external drainage is employed the patient must be told that the tube will have to stay in for months. Many patients, particularly women, object to this because they find it difficult to wear their clothing over this tubing even though the vertical part of the T tube is clamped off. It is a little messy, because a little mucoid secretion comes around all such external tubes that go into an internal cavity. However, prolonged T tube drainage in cases of mild degrees of chronic pancreatitis has been effective in relieving the severity of the pain and reducing its frequency in about 75 per cent of the cases.

The last procedure of value is splanchnicectomy. Before splanchnicectomy is undertaken, NOVOCAIN® injections should be performed. The only scientific way is to inject procaine hydrochloride into the left splanchnic nerve of patients when they are having an attack of pain. If pain is relieved (and it frequently is), then a little absolute alcohol can be injected with six months' relief of pain. If a patient gets six months' relief of pain and then continues to have pain after that, there are two alternatives: You can reinject the nerve or you can have your neurologic surgeon cut the left splanchnic major and minor trunks.

We have three minutes left and we just want to say a word about cancer of the pancreas. How frequently in cancer of the head of the pancreas producing jaundice in a patient in good condition, do you, Mr. Chamberlain, do a radical resection of the head of the pancreas, the so-called Whipple operation? How frequently should the less serious but the palliative procedure of biliary intestinal anastomosis, short-circuiting the bile around the obstructing head of the pancreas, be employed and what do you think are the indications for it?

MR. CHAMBERLAIN: In my country there has been a swing away from the Whipple operation, partly because of the poor long-term results, but I think probably the indication is going to be the very small, obviously resectable tumor.

There is just one point that I would like to make in treatment, which may or may not be familiar to you, and that is that in these jaundiced patients there has been a suggestion that the operation should be divided into two parts. I would like to put before you the possibility of a simple cholecystostomy. A cholecystostomy commits you to nothing because the patient gets over his jaundice, you can then re-explore your patient, and if you decide at that re-exploration that the patient is inoperable, you can detach the gallbladder and use it for an anastomosis, and if you decide that the operation is feasible, it still does not stop that procedure. I think that that type of drainage, if you are going to embark on a two stage procedure, has very definite points in its favor.

DR. WALTERS: I, on the other hand, would disagree with that. I did a good many, years ago, and because of the hydrohepatosis (the dilatation of the biliary ducts within the liver) and the loss of fluid and salt, which was considerable, I discontinued use of the preliminary cholecystostomy. I have found that the distended gallbladder can be safely anastomosed to the stomach or to a loop of jejunum, if you wish, or the distended common duct can be anastomosed to the duodenum, and then in six weeks or two months, preferably six weeks, the anastomosis can be detached easily if radical resection of the head of the pancreas is desired.

Lastly, we are both agreed on the advisability of radical resection of the ampullary lesion, are we not?

MR. CHAMBERLAIN: Yes. However, I was going to disagree with what you said a moment ago, but we haven't time—I will do it outside.

CLINICOPATHOLOGIC CONFERENCE

Report from Veterans Administration Hospital, Hines, Illinois

Report of Case

A 59 year old white woman was readmitted to the hospital on November 17, 1952 for treatment of severe diabetes. She had gangrene of the right foot. She had had diabetes for 26 years. Previous operations included tonsillectomy, appendectomy, hysterectomy for adenocarcinoma of the uterus in 1950, and amputation of the second, third, fourth and fifth toes of the left foot in 1951.

On admission, the patient's blood pressure was 166/94. Examination disclosed a draining sinus at the base of the second toe of the right foot and an abscess on the sole. X-rays of the foot revealed osteomyelitis of the heads of the first, second and fifth metatarsals. Vascular study showed excellent arterial circulation of the lower extremities.

On November 21, incision and drainage of the right plantar fascial abscess were carried out, and all five right toes were amputated through the metatarsal shafts.

On January 15, 1953, because of persistent cellulitis of the right foot, a guillotine amputation of the right leg was done, 3 in. above the malleoli. On January 28, it was necessary to reamputate the right leg 1½ in. above the previous level because of an open guillotine stump.

On May 2, the patient had a special medical examination and a diagnosis of hypertension, arteriosclerotic heart disease and generalized arteriosclerosis was made. On June 10, revision of the cicatrix of the amputation of the right leg was done, with removal of the hypertrophic scar on the end of the stump. By August, the stump had healed and the patient was ready to use her artificial limb.

Beginning in November 1953, the patient had eight separate attacks of abdominal distention and pain. Each attack lasted a few days and disappeared following the use of cathartics and the passage of flatus. Physical examination revealed the bowel sounds to be active and the abdomen tympanitic. Masses could not be felt. Palpation of the right flank and right side of the abdomen produced pain. The patient's temperature, pulse and respirations were normal. These complaints and findings persisted, and on January 28, 1954 she was seen by the Tumor Board because of the abdominal distention. The impression at that time was "intestinal obstruction, incomplete, recurring attacks, cause undetermined," and a further medical work-up was recommended.

On February 4, 1954, at which time the cause of the abdominal distention still was undetermined, the patient had surgical con-

sultation and an exploratory laparotomy was recommended. Operation on February 25 failed to reveal any obstruction. The extrahepatic biliary system was normal, and the liver was slightly reduced in size and gray in color. On March 5, a postoperative wound disruption was repaired. On April 11, the patient was seen by a consultant in ophthalmology, who found an incipient central right subcapsular cataract.

About June 10, the patient noted the onset of nausea and vomiting, and pain in the right upper quadrant developed, radiating to the right posterior midscapular region. Later she noted the passage of clay-colored stools, and jaundice developed and became progressively worse. Physical examination revealed only abdominal distention and pain. Prior to the onset of jaundice, the diabetes had been fairly well controlled with 75 to 85 units of regular insulin per day. Following the onset of jaundice, the patient's insulin requirements became less. She was unable to retain food, and it was necessary to maintain her on parenteral fluids.

On June 23, the common duct was explored and found to be patent. In order to insure biliary drainage, a T tube was placed in the common duct. At that time, the liver was small and gray and presented rounded margins. The subdiaphragmatic space was normal. A diagnosis of hepatitis was made at operation. On the twelfth postoperative day, pain developed in the right upper quadrant. It was aggravated by deep breathing. The patient began to have an intermittently elevated temperature—as high as 100 to 101° F. The drainage from the T tube decreased in quantity, her general condition deteriorated rapidly, and she became comatose. She died August 6, 1954, about six weeks after the exploratory operation.

The laboratory findings in this case are summarized in table 1.

Differential Diagnosis

DR. JOHN H. OLWIN: First, to emphasize the known pathologic findings in this case, the patient had been a diabetic for 26 years. She had osteomyelitis over a period of time, adenocarcinoma of the uterus (removed in 1950), and repeated amputations. She also had moderate hypertension and a cataract of the right eye.

It seemed to me that there were two problems here as to differential diagnosis. The first concerned an explanation for the attacks of abdominal pain. Appendicitis perhaps should be ruled out first. Her appendix had been removed some years previously. Of course, there could be adhesions, and the possibility of Meckel's diverticulum must be considered, but the picture is definitely that of an obstruction. The pain was intermittent, bowel sounds were active, abdominal distention developed later, and masses were not felt. She had pain and tenderness over the right side of the abdomen. Pulse, temperature and respirations were not abnormal. In instances of obstruction, the pulse rate rises very markedly, particularly with strangulation, but there could be obstruction without too much disturbance of pulse or temperature.

On February 19, 1954, the x-ray flat plate of the abdomen was read as "a partial bowel obstruction in the region of the proximal ileum, in the region of the left lower quadrant of the abdomen, probably on the basis of adhesions of the bowel in this region." This was a fairly clear-cut indication for an exploratory operation, which was done, but nothing was found. It seemed to me that the operation very definitely was indicated. The laboratory findings supporting obstruction, other than the x-ray report, are not important.

The diagnosis of kidney stones should be considered, although the pain in the right side certainly was not the picture of kidney stones. Urinalysis did not disclose any unusual findings, and there was nothing on the flat plate to suggest this disorder. I do not believe a urogram was made, but certainly the picture was not that of kidney stones.

Gallbladder disease also should be considered. However, the tenderness on the right side was not the type one usually finds in such cases, and a cholecystogram which was made later did not suggest gallbladder difficulty.

I think we certainly should consider the possibility of an amebic infection. Amebic

TABLE 1

LABORATORY FINDINGS

DATE	FINDINGS
11/17/52	Wassermann negative. Urinalysis: acid reaction, specific gravity 1.011; albumin, trace; sugar, positive; white blood cells, moderate; red blood cells, negative; casts, negative; acetone, negative. Red blood cells 3,650,000. Hemoglobin 70 per cent. White blood cells 11,200; polymorphonuclear leukocytes 62, lymphocytes 35, monocytes 2, eosinophils 1. Fasting blood sugar 103.3. Total protein 8.2; albumin-globulin ratio 5.3/2.9.
11/4/53	Urinalysis: sugar, trace; acetone, negative. Red blood cells 4,100,000. White blood cells 5600; polymorphonuclear leukocytes 70, lymphocytes 26, eosinophils 4. Fasting blood sugar 332. Blood amylase 26.2 (normal 20 to 40). Serum bilirubin 1.2.
12/3/53	Three stool specimens negative for parasites, ova, protozoa and occult blood.
1/28/54	Red blood cells 4,970,000. Hemoglobin 15 gm. White blood cells 7200; polymorphonuclear leukocytes 52, lymphocytes 38, eosinophils 10.
2/19/54	Red blood cells 4,400,000. Hemoglobin 13 gm. White blood cells 5000; polymorphonuclear leukocytes 63, lymphocytes 30, eosinophils 7. Platelets 240,000. Fasting blood sugar 363.
3/5/54	Nonprotein nitrogen 35.6. Carbon dioxide-combining power 60 per cent. Total protein 7.2.
6/5/54	Icteric index 41. Serum bilirubin 6.4. Van den Bergh: direct 7.2, indirect 10.4.
6/11/54	Urinalysis: urobilinogen, negative; bile, positive. Red blood cells 4,850,000. Hemoglobin 15 gm. White blood cells 4000. Blood amylase 22.7. Alkaline phosphatase 21.8. Blood cholesterol 370 (normal 140 to 230). Cholesterol esters 90. Total protein 7.3; albumin-globulin ratio 3.7/3.6.
6/17/54	Icteric index 108. Cephalin flocculation 4 plus. Prothrombin time 13 seconds (normal). Van den Bergh: direct 13.9, indirect 20.9. Thymol turbidity 20 units. Occult blood in stool 2 plus.
6/21/54	Icteric index 142. Amylase 40.5. Van den Bergh: direct 10.5. Urinalysis: urobilinogen, negative; bile, positive. Red blood cells 4,750,000. Hemoglobin 14 gm. White blood cells 6400. Fasting blood sugar 326 mg. per cent. Alkaline phosphatase 13.8. Nonprotein nitrogen 31. Blood calcium 10.0. Inorganic phosphorus 3.0. Total protein 6.8. Stool, occult blood 4 plus.
7/4/54	Total protein 6.9; albumin-globulin ratio 2.9/4. Icteric index 128. Alkaline phosphatase 6.7. Van den Bergh: direct 19.2, indirect 30.3. Thymol turbidity 20 units. Blood chlorides 591. Carbon dioxide-combining power 53 vol. per cent.
7/14/54	Total protein 6.2; albumin-globulin ratio 2.5/3.7. Icteric index 140. Alkaline phosphatase 8.0. Cephalin flocculation: 24 hours, 3 plus; 48 hours, 4 plus. Thymol turbidity 20 units. Stool: urobilinogen, 1 plus.
7/29/54	Red blood cells 2,150,000. White blood cells 8800; polymorphonuclear leukocytes 92, lymphocytes 8. Fasting blood sugar 258. Total protein 6.5; albumin-globulin ratio 2.1/4.4. Icteric index 128. Van den Bergh: direct 18, indirect 28.9. Thymol turbidity 15 units.
8/5/54	Total protein 5.8; albumin-globulin ratio 2.6/3.2. Icteric index 126. Van den Bergh: direct 16, indirect 28.8. Thymol turbidity 12. Carbon dioxide-combining power 42 vol. per cent. Fasting blood sugar 341.
Electrocardiograms	
10/8/53	Interpretation: abnormal; myocardial damage.
1/18/54	Interpretation: abnormal; consistent with residual of anterior wall infarction.
6/16/54	Interpretation: left axis deviation; consistent with transverse heart and left strain.
X-rays	
11/21/52	Chest: essentially negative.
11/21/52	Right foot: osteomyelitis of heads of first, second and fifth metatarsals.
10/26/53	Graham-Cole test. Impression: no evidence of biliary calculi; gallbladder concentrates the test material less satisfactorily than usual.
2/19/54	Gastrointestinal series. Impression: Findings suggest a partial bowel obstruction in region of the proximal ileum, in the region of the left lower quadrant of the abdomen, probably on the basis of adhesions of the bowel in this region; hypermobility of the small bowel.

dysentery in this country does not present the typical textbook picture we learned in school. Often it gives a very vague type of pain that comes on at times, may be regular, or may come and go. It is characterized often by distention, with generalized pain and passage of a lot of gas. However, examination of the stool in this case did not disclose any parasites. In fact, it did not show anything remarkable. On three separate occasions, blood was not found in the stool. However, we know that sometimes one does not find parasites in the stool in cases of amebic infection, and sometimes the patients get well if they are given treatment. I believe one of the things which might have been done was to try antiamebic therapy, which could have been given without danger.

The medical men suggest that there might have been something in the way of a segmental type of ileus found in diabetics. I tried to find something about this in the literature but was unable to, and I would be glad to have some illumination on this, as it certainly is a very interesting subject.

It would seem that those were the main points which might be considered. As we know, an explanation for these attacks was not found at surgery or during the rest of the clinical course. Apparently, recurrent adenocarcinoma was not found, although mention was not made of this.

The second problem concerns the picture of nausea, vomiting, pain in the right upper quadrant which radiated to the right posterior midscapular region, and the later appearance of clay-colored stools and jaundice in June 1954. You will recall that extrahepatic disease was not found at exploration in February. At that time the liver was slightly reduced in size and gray in color.

I should mention here a fact which should have been brought up earlier, and that is that this patient had four transfusions of blood and five transfusions of plasma over a period of three months prior to and during the last surgical procedure.

This last picture certainly suggests obstruction of the extrahepatic biliary system—obstruction of the common duct, particularly. She had the right upper quadrant pain radiating to the back, the clay-colored stools and jaundice. Consideration also should be given to obstruction of the biliary system because of a stone. Before the first exploration, a cholecystogram did not show evidence of biliary calculi and the gallbladder concentrated the test material in less time than normal. The patient's icteric index was rising steadily, and certainly one would expect that in obstruction of the biliary tract; the icteric index was 142 on June 21, two days before exploration.

Against this picture of common duct stone, however, are the findings which suggest liver disease on the basis of the laboratory examinations. One or two prothrombin determinations were normal. In an ordinary case of obstruction of the common duct, liver damage does not occur unless the obstruction persists over a period of time. Then the liver function begins to diminish. Along this line, we did some studies a few years ago, using the two stage prothrombin determination, on extrahepatic and intrahepatic liver disease, particularly with jaundice. We found that liver damage was indicated fairly well by the response of the prothrombin on the basis of the two stage test. This test proved much more sensitive than the Quick test. If a patient has a 25 per cent prothrombin and is given vitamin K over a period of several days, the prothrombin will come back to normal within 48 to 72 hours if the difficulty is on the basis of an obstruction—if, that is, the obstruction is of not more than three, four or five weeks' duration. If, on the other hand, the patient has hepatitis, there will be perhaps a 60 or 70 per cent prothrombin; with vitamin K therapy it will come up 10 to 15 points, but as long as hepatitis persists it does not come up much further. If cirrhosis is present, the prothrombin may come up from 45 to 60 per cent under treatment, if it is not a severe case. In severe cirrhosis it will be raised only a few points. When a prothrombin level does not respond, we usually suspect severe liver damage.

The possibility of cirrhosis cannot be ruled out. The patient's history does not support it, nor did the examination of the liver at exploration. However, she did have jaundice,

and we therefore must consider this possibility. Usually there is some liver enlargement and we can feel it, but in the atrophic state there may be a small liver. The various liver function tests will show some liver damage.

Ordinarily, in the presence of subacute yellow atrophy, one finds a shrunken liver. Jaundice usually becomes severe, and as a rule the liver function diminishes markedly. One also finds signs of prothrombin insufficiency of the skin—big blotches of hemorrhage—and bleeding from the nose. Usually these are noted in the late stages shortly before death. I think we should certainly consider the possibility of subacute yellow atrophy.

One other thing we should consider is hepatitis. I think that was the diagnosis considered to be most likely before the patient died. It was the diagnosis made at operation during the last exploratory procedure, and certainly there is much to support it—jaundice, pain in the right upper quadrant, diminished liver function with hepatocellular damage as shown by a number of laboratory tests. The liver might or might not be palpable in hepatitis, depending on the severity of the condition.

One other thing might well have been done—a biopsy of the liver at the time of the last exploration. I believe a biopsy is considered the best liver function test, and usually it can be done without danger of hemorrhage. Absorbent sponges placed in the defect will stop any hemorrhage.

It therefore seems that hepatitis is the most likely diagnosis. The etiologic basis for this might be a homologous serum reaction from the several transfusions of whole blood and plasma which the patient received. During the war, many of you saw cases of severe hepatitis, sometimes fatal, due to yellow fever vaccine or some virus in the blood or plasma used in the manufacture of the vaccine.

I would like to mention, too, the possibility of a subphrenic abscess. This probably would have been seen clearly at exploration, but it should be considered because of the pain in the right upper quadrant, the spiking temperature and the elevated diaphragm noted on x-ray examination.

I suppose that at autopsy you might find, in addition to hepatitis, fatty infiltration of the liver because of long-standing diabetes and generalized arteriosclerosis. There may or may not be changes in the islands of Langerhans, certain amyloid changes in the liver and spleen, subphrenic abscess, and yellow tissue due to bile pigment, and there will be absence of appendix, uterus and lower extremities, and cataract of the right eye.

Postmortem Findings

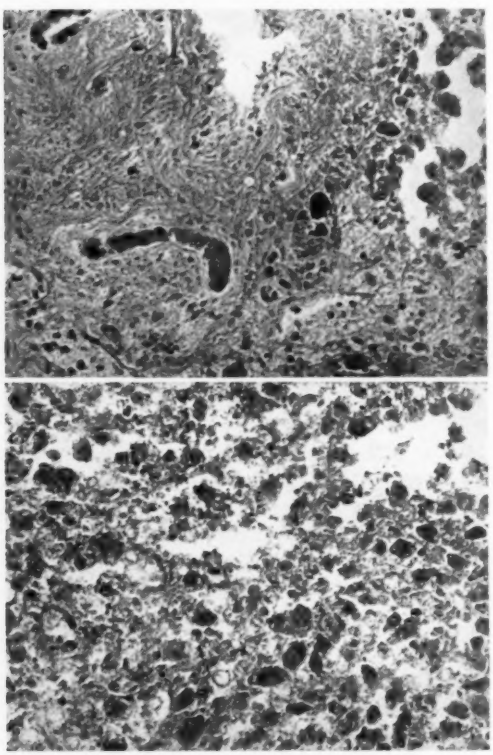
Examination disclosed intense jaundice. The abdomen was distended. The right leg had been amputated approximately 6 in. below the knee, and all the small toes of the left foot had been amputated. Hair was gray, pupils were equal, and sclerae jaundiced. The mouth was edentulous. Three vertical incision scars were noted on the abdominal wall—one a right rectus, one a right paramesial, and one a suprapubic. The peripheral vessels were thickened and firm; peripheral lymph nodes were not enlarged.

Examination of the abdomen showed a pad of fat 2.5 cm. thick. The lower portion of the peritoneal cavity was smooth. There were adhesions to the abdominal wall, and a biliary fistula extended to the right upper rectus scar. Around the fistulous tract, four or five small subcutaneous abscesses were noted; these were filled with yellowish-gray pus.

The liver was small, weighing 1,250 gm., greenish-yellow in color, soft and mushy in consistency, and easily crushed between the finger and thumb. Grossly, it had the appearance of a spleen. Cut section disclosed complete loss of markings and a soft, granular, grayish-yellow parenchyma. Over the dome of the liver and under the diaphragm there was about 700 cc. of yellowish pus. Microscopic examination of the liver revealed complete necrosis of all liver cells except for a few visible cells around the periphery of the lobule. Outlines of many of the cells were still visible, and the architectural stroma was not disturbed. There was hemorrhage beneath the capsule, and some scattered leukocytes, mostly polymorphonuclear leukocytes, were noted throughout the section.

The spleen was enlarged slightly, weighing

FIGURE 1. Gross appearance of liver in serum hepatitis.



FIGURES 2 and 3. Microsections of the liver in serum hepatitis, showing necrosis and infiltration.

340 gm., reddish-gray and hyperplastic. The pancreas was grossly normal, and microscopically it did not show any particular lesions. The kidneys were enlarged, the combined weight being 390 gm. Both were greenish-yellow and swollen, and the capsule was tight. Microscopically there was quite marked swelling and degeneration of the tubular epithelium, especially of the lower nephron. Bile pigment filled the lumen throughout. Many of the glomeruli were hyalinized.

There was absence of the uterus, ovaries and fallopian tubes. The heart and vascular system showed advanced arteriosclerosis.

Anatomic Diagnosis

1. Hepatitis, infectious type, with nonobstructive jaundice.
2. Subphrenic abscess, right.
3. Multiple abscesses, abdominal wall.
4. Biliary fistula.
5. Absence (acquired) of uterus, adnexa, right leg, left small toes.
6. Arteriosclerosis, generalized.

Discussion

PATHOLOGIST: It seems to me that this is a pretty good case of acute hepatitis, probably serum hepatitis. You will notice that this is one case in which we are able to demonstrate the lesion in diabetes mellitus. Very rarely can we find atrophy and fibrosis of the islands of Langerhans and fatty substitution in the pancreas. I think the kidney lesion that Dr. Zivin suggested probably represents the Wilson syndrome in diabetes. We have had three or four of these hepatitis cases here in the past three or four months. I think this is a very classic histologic picture of acute hepatitis. The history of transfusions of blood and plasma, which is not included in the protocol that you have, was dug out of the chart later, and should have been included.

DR. SIMON ZIVIN: I would like to ask if anyone has information on the behavior of segmental lesion of the bowel in diabetes mellitus. Just recently I noticed some suggestions about this in one of the consultant's records.

DR. IRVING ARIEL: A number of workers at Montefiore Hospital in New York have been

studying this question. They believe that the same type of arteriolar disease which is seen in the kidney with the advanced vascular changes of diabetes is also found in the intestine and may form involved segments which are very similar to regional ileitis.

DR. WILLIAM METCALF: A few factors should be noted which may help in distinguishing between infectious hepatitis and serum jaundice. In the former, the incubation period is between 14 and 35 days; in the latter, it is from two to four months. The course of infectious hepatitis is fairly benign and the mortality rate low. As a matter of fact, about 95 per cent of the men who had this disease in the Army returned to duty. On the other hand, serum jaundice runs a much more severe course and the mortality rate is higher.

It should be noted, too, that these diseases are not necessarily related. It was discovered during the war that soldiers who had recovered from infectious hepatitis were not immune to serum jaundice. Many who had had infectious hepatitis contracted serum jaundice when inoculated with its virus in yellow fever vaccine or in plasma or from a contaminated needle or syringe.

In practice, this implies that a donor should not be used who has a history of jaundice within a year or who has been exposed to infectious hepatitis or who recently has had a transfusion of blood or plasma. Also, plasma pools should be kept small so that if one is contaminated with the virus the number of recipients to whom the disease might be transmitted would also be small.

PATHOLOGIST: We had three or four of these cases, and I think they all gave very similar histories of transfusions over a period of three, four or up to six months.

Dr. H. Mason Morfit, Associate Professor of Surgery, University of Colorado Medical School, and Director, The Bonfils Tumor Clinic, Denver

CANCER OF THE MOUTH AND NECK

Curability of any form of cancer today depends largely on the stage of disease at which definitive treatment is begun. Cancer of the mouth is an accessible form of cancer and, therefore, provides a fair chance of early discovery and a favorable prognosis. Dr. Morfit will discuss the symptoms and manifestations of the disease and its management.

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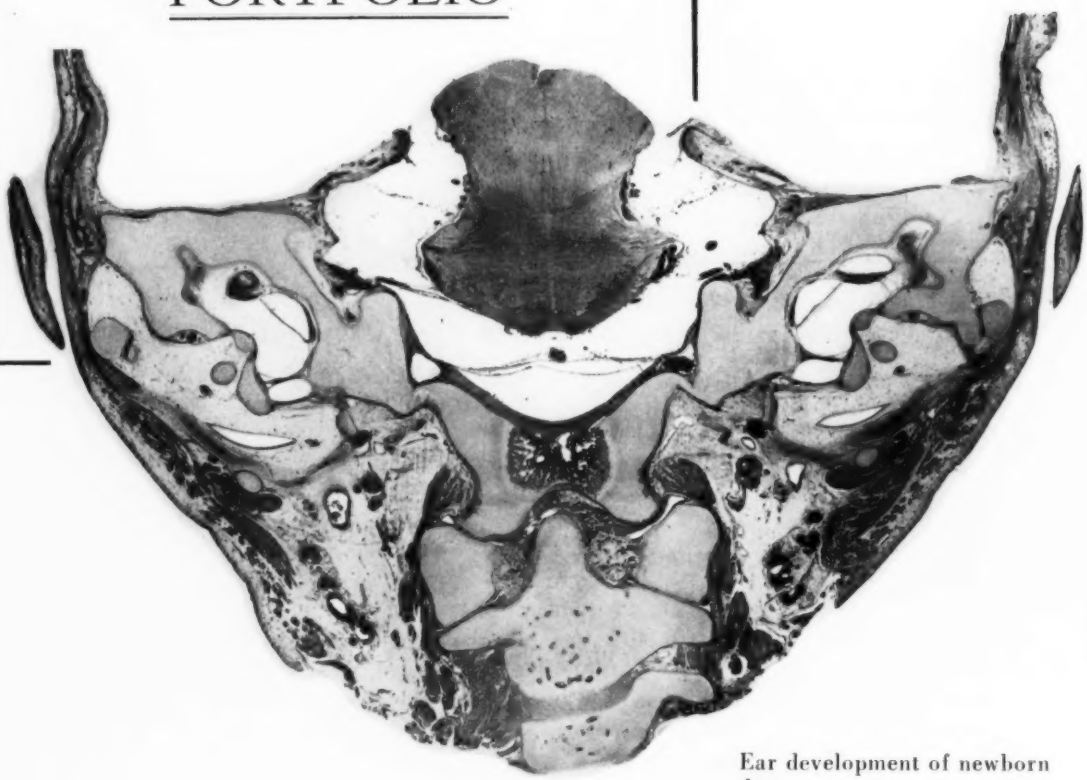
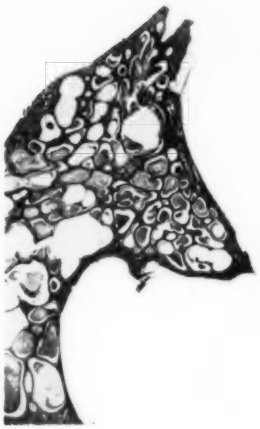


Ear development of newborn dog.

Maria Ikenberg, FBPA, University of Illinois College of Medicine, Chicago.



Humorously entitled "Doggone human acute mastoiditis," from Dr. M. J. Tamari collection.

Maria Ikenberg, FBPA, University of Illinois College of Medicine, Chicago.

THREE things every medical writer owes to his readers: (1) familiarity with his subject, (2) a more than casual acquaintance with the language, and (3) a grasp of the purpose and scope of an illustration.

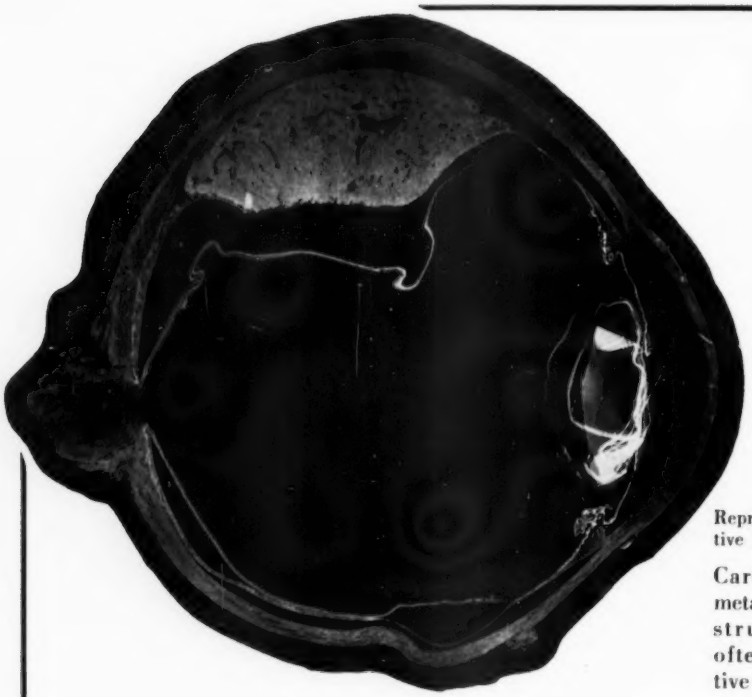
The platitudes to the effect that "one picture is worth a thousand words" and that "the camera never lies" have been shot full of holes, and the medical author will do well not to rely on them. If it should turn out that the replaced thousand words are not true then the photograph becomes a giant whopper. Furthermore, if a photograph equals a thousand words, then 10 photographs will equal 10 thousand words, which could become tiresome.

The camera is by nature truthful, but any good photographer can lead it into some very fancy double talk; however, what may at first seem a distortion of the face of nature can often be turned to good account in medical illustration. Far from being misleading, it can accentuate important details, suppressing the rest by exaggerating contrast or perspective. Such treatment is not the result of a happy accident, but is under the complete control of the photographer. Through countless combinations of lenses, filters, films, developers, papers, lights and angles, he can emphasize structures and textures to meet the requirements of the engraver. Examples of such treatment are the accompanying photographs, made by infrared light, of the superficial veins of the chest and the leg. These pictures, almost diagrammatic in clarity, can only be made by photographic methods. By contrast, but also demanding photographic treatment, note the characteristic texture of the skin brought out in the illustrations of breast cancer.

Medical editors everywhere are keenly aware of the high quality of photographic illustration now available and are very properly demanding it for their journals.

ARTHUR W. PROETZ, M.D., F.B.P.A.
Editorial Board Member, *Journal of the Biological Photographic Association*

Because, as Dr. Proetz has pointed out, illustration is so important to medical publishing, POSTGRADUATE MEDICINE is pleased to offer these selected examples of outstanding medical photography by members of the Biological Photographic Association. The medical photographer, working closely with the medical author, contributes greatly to the effective presentation of the author's material. The program of B.P.A., which is the medical photographers' own professional association, has as its goal the establishing and maintaining of the highest standards for medical photography of all types, and the services of its members can be profitably utilized by all physician-writers in preparing material for publication.



Reproduced from a negative print.

Carcinoma of lung, metastatic to eye. More structural detail is often seen in a negative print.

Reproduced from a positive print.

F. C. Breckenridge, Baylor University College of Medicine, Houston.

Photomicrograph—Nematode-hookworm biting wall of small intestine.

Photomicrograph—Acute myeloblastic leukemia.

Stella Zimmer, FBPA, and Louis Georgiana, State University of New York at Syracuse.

Ear lobe returned after traumatic avulsion.

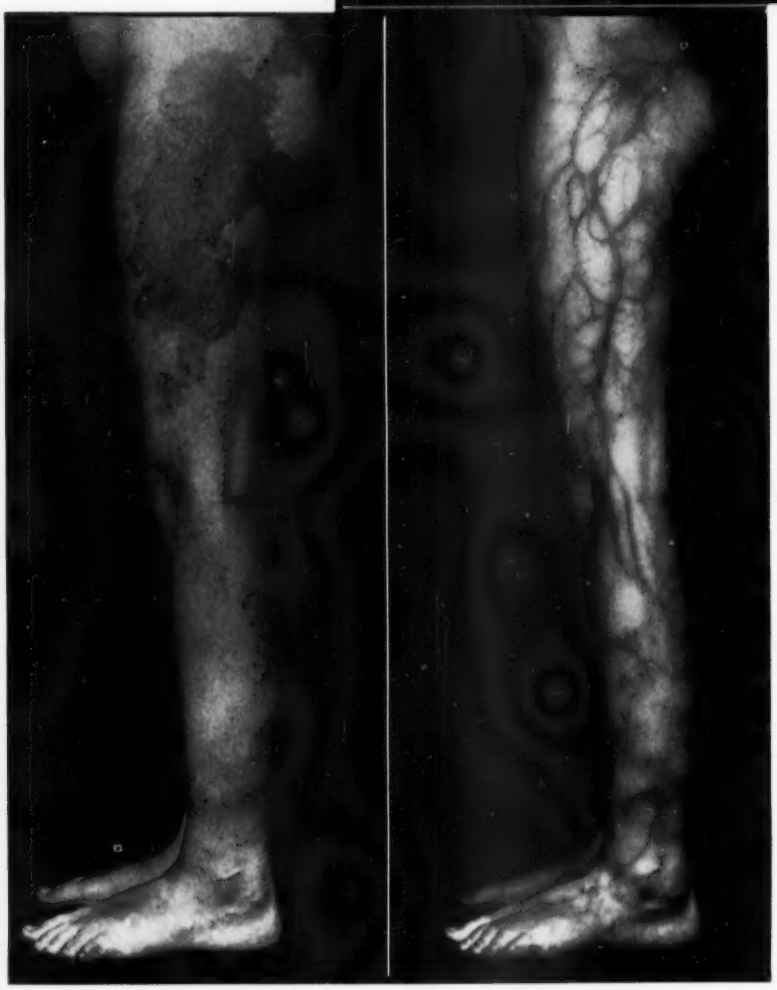
Lloyd Matlovsky, University of Southern California, Los Angeles General Hospital, Los Angeles.

Early cancer of the breast.

Lloyd Matlovsky, University of Southern California, Los Angeles General Hospital, Los Angeles.

▲ Infrared photograph showing considerable engorgement of superficial venous circulation on left side. No unusual venous pattern seen when viewed by visible radiation.

Lewis W. Koster, Columbia University, New York.



◀ Cavernous capillary hemangioma of the left leg extending from the lumbar region to the toes. This congenital defect is characterized by the extensive venous pattern seen in this infrared photograph.

Lardner A. Coffey, FBPA, Mayo Clinic, Rochester, Minnesota.

Erythroblastosis Fetalis

NEVA M. ABELSON*

University of Pennsylvania School of Medicine, Philadelphia

NEVA M. ABELSON

ONE of the most interesting recent developments in the field of the blood groups has been the demonstration of a correlation between the presence of ABO factors in the blood and the incidence of certain diseases.[1,2] At the same time, there has been renewed and intensive investigation of the relation between these factors and erythroblastosis fetalis.[3-7] As a result, experts in the field believe that the two most common (and perhaps about equally common) etiologic agents in hemolytic disease of the newborn are the Rh factor and either the ABO factors or a very closely related antigen.

Although Rh and ABO erythroblastosis fetalis are both hemolytic diseases of the newborn, they are in many respects dissimilar. In Rh erythroblastosis fetalis there is a clear-cut serologic pattern. Our case material indicates that about one in 17 Rh-negative women becomes Rh sensitized through pregnancy and that in practically every such case maternal serum antibodies can be found. Incompatibility can be confirmed by demonstrating the coating of fetal red blood cells by transplacentally acquired antibody (positive direct Coombs test). In ABO erythroblastosis fetalis, the serologist can offer far less help. About one-fourth of all pregnancies are heterospecific; that is, they involve babies whose A or B blood groups are incompatible with the mother's serum. Most of these infants escape apparent hemolytic disease. In fact, when erythroblastosis fetalis occurs, the mother usually is group O and the baby group A. Therefore, it is apparent that routine antenatal blood group screening tests are of limited diagnostic value. Further serologic tests which offer only presumptive evidence of disease, but which are worthy of consideration, are:

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Determining the height of the maternal anti-A or anti-B titer—Anti-A saline titers above 1:128 and anti-B titers above 1:64 may be held suspect, but the presence of lower titers does not exclude the possibility of ABO hemolytic disease, and the children of mothers with high titers often are unaffected.

Search for maternal "immune" anti-A or anti-B agglutinins—In many cases of ABO erythroblastosis fetalis the maternal anti-A or anti-B titer is significantly higher in protein diluents than in saline. These agglutinins are difficult to neutralize with specific polysaccharides. However, many women with immune agglutinins give birth to normal babies, and infants with ABO hemolytic disease are born to women without immune antibodies.

Search for maternal serum hemolysins for fetal red blood cells—It has been suggested that in doubtful cases unless the maternal serum hemolyzes the infant's blood cells (not adult cells) in the presence of human complement, A-O erythroblastosis fetalis is unlikely.[8]

Direct antiglobulin (Coombs) test—In my experience the direct antiglobulin test is seldom, if ever, strongly positive in ABO erythroblastosis fetalis. If the test is done carefully, however, and read carefully, weakly positive results often are obtained.

Absorption and elution tests—It has been known for years that A cells, after exposure to group O serum and later washing, may yield an eluate capable of agglutinating both A and B cells. By the same token, when group O serum is absorbed with group A or group B cells, titers of both anti-A and anti-B may be reduced. Some serologists believe this phenomenon is due to linkage of alpha and beta agglutinins in group O serum; others believe that group O serum contains a third antibody, anti-C (not related to the Rh system). The whole subject has been reopened recently, and further information is available in the literature.[9-12] As yet, however, the investigations have not led to a convenient or accurate diagnostic test.

From the foregoing, it may be inferred that at present antenatal tests for ABO erythroblastosis fetalis are of little more than academic interest, and this inference is correct. Indeed, such laboratory examinations all too often cause unnecessary apprehension and expense. Nor do they appear to be essential in safeguarding mother and child. ABO erythroblastosis fetalis is usually a mild disease. Hydrops fetalis and intra-uterine death do not occur often enough to cause concern, and profound anemia on the first day of life is seldom encountered. Therefore, diagnosis and treatment may be deferred until supportive hematologic, chemical and clinical data are available.

The various Rh determinations, on the other hand, are invaluable. I believe that all prospective parents should be tested for Rh incompatibility and that in the occasional case where the husband and wife both appear to be Rh negative, special tests for the presence of the D^u variant should be performed. In our clinics Rh-negative prenatal patients are also tested for presence of the D^u factor, and about 15 per cent of supposed D-negative patients have proved to be D^u positive. These patients are followed antenatally in a special clinic, but in only two cases have we seen abnormal agglutinins develop. In neither case did clinical erythroblastosis fetalis appear. I also recommend that every Rh-negative woman, regardless of parity, have at least three Rh antibody tests during pregnancy, one soon after 16 weeks, another at 28 to 32 weeks and a third at 32 to 36 weeks. The first test provides a base line from which to judge the subsequent course of the titer. The second test serves to put the parents and the obstetrician on guard for a possible intra-uterine death, and the third test will pick up most (but not quite all) serious sensitizations which may occur late in pregnancy. In the event antibodies appear, I also recommend that the husband's Rh subgroup be determined for presumptive evidence of homozygosis or heterozygosis for D. If the titer is changing, more frequent antibody tests may be indicated. Repeated tests are unnecessary in known highly sensitized cases unless the husband is heterozygous and the physician is searching for an indication of whether the fetus involved is Rh positive or Rh negative. Frequent tests also are unnecessary in the nonsensitized case. However, it is hazardous for a number of reasons to postpone serologic ex-

aminations until the last three or four weeks of pregnancy. These reasons are:

Intra-uterine death may occur at any time after the twenty-fourth week and in aggravated cases it sometimes occurs even earlier. This event is accompanied by three potential dangers to the mother which must be regarded seriously. The first (and it usually does not receive the consideration it deserves) is that of the emotional shock which may follow. The good physician does not allow parents to face tragedy without adequate preparation. The second is the possibility of rapidly developing polyhydramnios with lowering of maternal serum proteins. The third is serious hemorrhage resulting from disturbance of coagulation factors.

Rh-sensitization may be complicated by spontaneous premature delivery. Time is of the essence in the successful treatment of the premature erythroblastotic infant and proper safeguards may mean the difference between success and failure.

There is reason to believe that in the occasional, properly selected case interruption of the pregnancy at the thirty-seventh or thirty-eighth week may be lifesaving. Serial anti-Rh titers may be helpful in selecting these cases.

Opinion as to the value of early delivery in cases where hemolytic disease of the newborn is likely to occur has changed over the course of the years. Originally it was believed that removing the baby from the action of maternal antibodies was desirable. In a few cases this was perhaps true, but in most instances early delivery was not beneficial. It soon became apparent that an undue proportion of premature infants survived with central nervous system sequelae (kernicterus). The pendulum then swung to the opposite direction, and until recently it was the consensus that early delivery was contraindicated. In the last two or three years the success of exchange transfusion in preventing kernicterus has led to such modification of opinion that in a few hospitals all Rh-sensitized pregnant women are delivered if possible at 37 or 38 weeks.

At present, early delivery in ABO erythroblastosis fetalis is still not indicated for the following reasons:

The disease usually does not lead to stillbirth or even to neonatal death. Its main hazard is kernicterus, which apparently is not an intra-uterine disease and which occurs oftener in premature than in full-term infants.

There is poor correlation between the maternal reproductive history and prognosis for the baby. In fact, the first baby is affected as commonly as are subsequent infants.

There is poor correlation between serologic findings and prognosis.

There is no convenient serologic test for homozygosis, and family study may not resolve this question.

I believe also that early delivery is not indicated in cases of Rh-sensitization in the following circumstances:

When the husband is heterozygous and there is no indication from the maternal anti-Rh titer that the infant is Rh positive. In many cases of Rh-sensitization the titer remains stationary even when the baby's blood type is incompatible. In other cases there may be a slight anamnestic reaction (one- or twofold) when the baby is Rh negative. In these circumstances it is impossible to predict the Rh type of the infant, and one must assume there is a 50-50 chance that it will be normal.

When there has been no previous affected infant and the maternal anti-Rh titer is under 1:64. In such cases, the outcome is likely to be quite good.

When the disease has been mild in previous affected babies and the anti-Rh titer has not changed significantly. In general (and of course there are exceptions) a mother who has had one or more mildly affected babies will tend to continue to have mildly affected children. For some years I have followed a family where 13 erythroblastotic infants were born to the same mother. Eleven of these infants recovered without sequelae, one (the sixth child) survived with cerebral palsy, and one (the eleventh) was stillborn. More recently, since the effectiveness of exchange transfusion was demonstrated, we have followed a number of similar, though less striking, cases and have observed the tendency toward repetition of the disease pattern established in the first affected baby.

Premature delivery may be considered:

When the husband is homozygous, the titer is 1:64 or over, and there is a history of previous incompatible blood injection. In a high percentage of these cases the infant is severely affected.

When the husband is homozygous, the titer is high (above 1:64) and there have been no previous mildly affected babies. These criteria are most useful in the case of a first affected baby, but may apply also when the family is small and the reproductive history is poor.

When there have been no previous affected children and the anti-Rh titer has clearly risen during pregnancy to 1:64 or more. In this event it is immaterial whether the husband is homozygous or heterozygous. Sometimes high saline titers produce only mild disease, but the chances of late stillbirth of a first affected baby when the titer is high are fairly great. The following case will serve as an example:

Case 1—Mrs. G.'s first baby was normal and there was no history of blood transfusion. Her husband was C-positive, D-positive, E-negative and c-positive (presumably heterozygous). At the twelfth week of pregnancy and again at the twenty-second week of pregnancy, no Rh antibodies were detectable. At the thirty-fourth week, however, the anti-D titer was 1:4 in saline and 1:64 in albumin. The possible value of early delivery was considered but rejected because there had been no previous erythroblastotic babies and because the husband was heterozygous. At term the antibody titer had disappeared in saline and had increased to 1:1024 in albumin. The infant was so severely affected at birth that despite immediate and repeated exchange transfusions he lived less than 24 hours. This was clearly a case where it could have been predicted that the infant would be Rh positive and where delivery two or three weeks before term might have been lifesaving.

When there has been a significant change in the character of the anti-Rh titer. The expert serologist should be consulted on this point. In general, the development of prozones in saline or in albumin indicates increased sensitization. The same is true when a saline titer disappears, apparently yielding to "blocking" antibodies. In such event, the titer in albumin, with enzyme-treated cells, or by indirect Coombs test will be as high as or higher than the previous saline titer. The case just cited is a good example.

When the anti-Rh titer has fallen significantly (twofold or more). This usually happens in severely sensitized cases, and the prognosis for the infant is poor.

When conditions are such that if delivery is spontaneous there may be undue delay in treating the infant. This applies to mothers living in isolated communities and to cases where compatible blood is not available on short notice. The following case illustrates both points:

Case 2—Mrs. M. was Rh positive (CDe/CDe) and her husband was Rh negative. When the second baby developed jaundice shortly after birth the attending pediatrician assumed the laboratory had made a clerical error in reporting the Rh tests and treated the infant with small transfusions of Rh-negative blood. The baby died of kernicterus at 48 hours. The parents lived in a small community about 60 miles away. When Mrs. M. reported for prenatal care early in her third pregnancy, her anti-c titer was 1:64 in saline and 1:128 in albumin. At the thirty-seventh week of pregnancy the titer was negative in saline and 1:128 in albumin. Labor was induced at this time. The infant required two exchange transfusions, one performed immediately after birth and the other at 24 hours. The baby has developed normally and at three years has no detectable neurologic sequelae. Here is a case where if labor had been allowed to occur spontaneously invaluable time might have been lost in getting the baby to a hospital and in finding compatible blood.

Any case of erythroblastosis fetalis potentially so severe as to warrant early delivery should be in the hands of physicians experienced in treating the disease, and no surgical intervention should be contemplated without investigating clotting factors in maternal blood. Fibrinogen now is available for treating obstetric hemorrhage which otherwise cannot be controlled. In the event early delivery is elected, the pregnancy should have reached

the thirty-seventh week and the infant should be estimated to weigh 6 lb. Our statistics indicate that 98 per cent of our adequately treated full-term erythroblastotic babies have lived and developed normally, whereas 80 per cent of our adequately treated premature erythroblastotic infants have lived and developed normally.

Aside from early delivery the physician is sometimes called on to consider therapeutic abortion in cases of fetal-maternal incompatibility. From what has been said already it is clear that this measure merits no consideration in cases of ABO incompatibility. Previous occurrence of intra-uterine death or kernicterus of the infant and afibrinogenemia, polyhydramnios or psychiatric disturbances in the mother must be taken into account in considering cases of Rh-sensitization, but in view of the variability of the disease and the recently improved methods of treatment, therapeutic abortion for Rh-sensitization alone appears difficult to justify.

Diagnosis of Rh erythroblastosis fetalis is relatively easy, but ABO cases are puzzling. In addition to serum bilirubin determinations, the following laboratory tests are helpful.

Search for spherocytes—Spherocytosis usually is present.

Tests of osmotic fragility—Osmotic fragility in sodium chloride solution may be increased. The mechanical fragility also usually is increased.

Search for reticulocytes—As a rule, reticulosis is present. The count often is 10 to 20 per cent and may be as high as 30 per cent.

Search for anemia—As a rule, mild or moderate anemia is observed. Very low red cell counts are not often seen.

Clinical signs, with one important exception, are not striking. The exception is jaundice, which usually appears within the first 24 hours. It is not often severe, and tends to clear rapidly. Serum bilirubin levels are above those of the average normal infant. Since jaundice is the most reliable clinical sign and since the prognosis is poorer in infants with high bilirubin levels, "jaundice rounds" are conducted in well-run nurseries. Any baby with an unusual degree of jaundice in the first 24 to 36 hours of life should have immediate, expert pediatric care. It is now agreed that erythroblastotic infants who develop high bilirubin levels in the first week of life are more likely to have neurologic sequelae than are the infants whose jaundice is less severe. This observation holds for all cases of hemolytic disease of the newborn, regardless of the blood group system involved. Therefore, treatment is aimed at preventing or correcting hyperbilirubinemia.

In ABO cases, it is usually safe to defer treatment until serial serum bilirubin levels indicate that a peak of 20 mg. per 100 ml. within the first three to five days of life may be expected. If the maternal history is poor, however, and the cord bilirubin level is over 3.5 mg. per 100 ml., it may be desirable to treat the infant immediately. It is difficult, without experience, to predict when the serum bilirubin level may reach its peak and when it may be expected to fall. In general, a value over 10 mg. per 100 ml. in the first 12 hours of life is regarded as dangerous, as is a level over 15 mg. per 100 ml. in the first 24 hours of life. Usually, bilirubinemia reaches a peak in the full-term infant within the first 72 hours of life; in the premature infant the peak may be reached as late as the fifth or even the seventh day.

All babies with ABO erythroblastosis fetalis whose serum bilirubins reach, or threaten to reach, 20 mg. per 100 ml. should have the benefit of exchange transfusion. No doubt using this criterion will lead to unnecessary treatment of many babies, but until we have better methods of assessing severity of disease there can be no choice. For although intrauterine ABO erythroblastosis fetalis is mild, the chances of neurologic involvement cannot be disregarded. Without good diagnostic criteria, accurate appraisal of the incidence of the complication is impossible, but it is agreed that the risk of kernicterus in jaundiced babies far outweighs the minimal dangers involved in the treatment. Freshly drawn group O blood is used for exchange transfusion, and we usually exchange twice the infant's estimated blood volume; in other words, the usual transfusion is 500 ml. We have not found it neces-

sary to add A and B specific substances or to remove a portion of the plasma. Occasionally, a second exchange transfusion is necessary to control the bilirubin level, but multiple transfusions are given far less frequently than in cases of Rh incompatibility. Booster transfusions are not often necessary after the original replacement procedure.

In cases where there has been a slowly falling blood cell count without hyperbilirubinemia, it may be necessary to correct anemia (red cell count of 2.5 million or less) by transfusion of packed group O cells (5 ml. per pound of body weight). Babies with ABO erythroblastosis fetalis rarely offer problems other than hyperbilirubinemia and anemia.

The diagnosis of Rh erythroblastosis fetalis is made on the serologic grounds already mentioned, and on the basis of evidence of red cell destruction in the infant. By no means do all Rh-positive babies with positive direct Coombs tests have significant clinical disease; in fact, such cases have been estimated to comprise between one-fourth and one-third of the total. This means that not all Rh-positive babies born to Rh-sensitized women are affected. In our laboratory we have found that titers which have never gone beyond 1:4 are unlikely to cause trouble. Likewise, antibodies detectable only by the sensitive enzyme-treated cell test are usually benign. On the other hand, I have not seen a proved case of Rh erythroblastosis fetalis where the direct Coombs test was negative.

Though mild cases occur, most infants with Rh erythroblastosis fetalis are more severely affected than those with ABO disease. Many are born dead or die in the first few hours of life, presumably of heart-failure. In the past two years, we have been successful in treating a number of cases which formerly would have been considered hopeless. These very sick infants require meticulous care. Immediate exchange transfusion is essential, and usually must be followed by re-exchange transfusion at suitable intervals. This treatment serves at least three purposes in such cases: correction of profound anemia, reduction of blood volume and control of blood pigments. About two years ago we established a technic of spectrophotometric examination of plasma pigments in erythroblastosis fetalis.[13,14] We have found this method invaluable in treating all our cases, and particularly in determining the need for re-exchange transfusion in babies born with severe anemia, edema, hepatosplenomegaly, high nucleated red cell counts and hemorrhagic tendencies. A case history illustrates points in managing such infants.

Case 3—Baby J. was a 9 lb. 11 oz. boy admitted at age five and one-half hours. His mother had had several Rh-incompatible blood transfusions and her antibody titer was 1:2048 in albumin. The initial red blood cell count was 2.2 million; the white cell count was 45,000 with 438 nucleated red cells per 100 white cells. The baby was moderately edematous. On admission he was lethargic, cyanotic and in obvious respiratory distress. Rales were heard throughout the lung fields. Numerous generalized petechiae were present. The spleen and liver were at the iliac crests; the Moro reflex was poor. The spectrophotometric absorption curve obtained on examination of the blood plasma is shown in figure 1 (top curve). An exchange transfusion of 1000 ml. was performed immediately. Two milliliters of 10 per cent calcium gluconate was given after each 100 ml. of blood transfused. At the end of the procedure the rales had diminished and the infant seemed a little more alert. At age 14 hours the blood pigments were as shown in the second absorption curve in figure 1, and an exchange transfusion (560 ml. out and 520 ml. in) was given. At 32 hours the blood pigments had returned to levels we considered unsafe, and a third exchange (525 ml. out, 500 ml. in) was done. At 56 hours the blood pigments were again at nearly the same levels as at 14 hours; therefore a fourth exchange of 480 ml. was carried out. The blood pigments then stabilized at the levels shown in the lowest absorption curve. The infant was kept in a high oxygen atmosphere with high humidity. Feedings were not started until respiration was satisfactory. On the fourth day the weight had fallen to 7 lb. 15 oz., and the baby appeared less lethargic. Subsequent improvement was rapid, and he was discharged on the ninth day. At that time

he had a systolic heart murmur which persisted for nearly three months. At two years he appears normal; we can detect no sign of brain damage.

It will be noted that a 40 ml. deficit was established in the second transfusion and a 25 ml. deficit in the third. This is good practice in cases with respiratory distress and high venous pressures. The importance of an atmosphere high in humidity and oxygen should be stressed. In this case there was no abnormality of blood chemistry, but if a baby so severely affected does not do well, the potassium, sodium, chloride and carbon dioxide levels should be determined. We have also occasionally seen abnormal calcium and nonprotein nitrogen levels.

It is hard to treat such severe cases without following the heme pigments closely, but it is helpful to know that the first 500 ml. exchange transfusion should not be expected to control the blood pigments for more than six hours. The second transfusion of 500 ml. may control them for about 12 hours; the third, for a little more than 24 hours. We consider a case to be controlled when the net optical density of the heme pigments in a 1:50 plasma dilution is less than 0.100. The formula for calculating this value may be found in reference 17. I believe it unsafe to allow the serum bilirubin to rise to more than 15 mg. per 100 ml. in these cases.

Choice of treatment for infants not so severely affected may be more difficult. The physician is too often tempted to wait a while to see if transfusion is really necessary. I am frequently asked to give criteria for exchange transfusion; one should seek, rather, to justify withholding the treatment. By following blood pigments carefully and frequently, we have been able to reduce the number of infants given exchange transfusion on our service to 70 per cent of the total number admitted. However, these laboratory procedures are too time-consuming for the busy practitioner. It is easier and safer, therefore, to elect exchange transfusion for all infants with Rh erythroblastosis fetalis, with these exceptions:

Low maternal titer and good reproductive history.

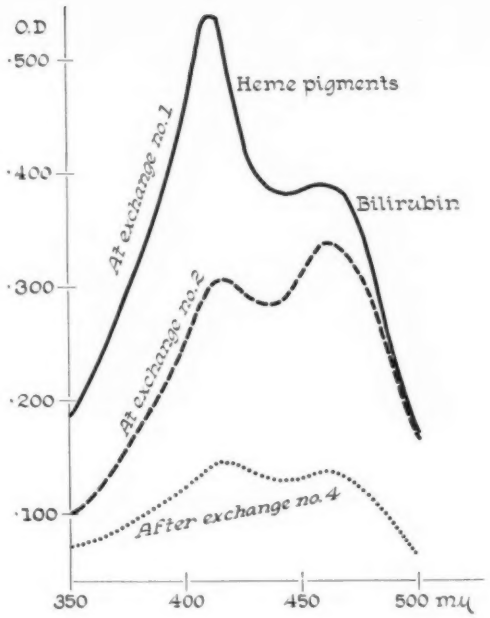

FIGURE 1. Continuous spectrophotometric absorption curves of 1:50 dilutions of plasma from baby with severe erythroblastosis fetalis.

Full-term infant (by date and weight).

Initial hemoglobin value of at least 15 gm. per 100 ml.

Serum bilirubin close to normal newborn limits, which are 2.5 mg. per 100 ml. at birth, 6.0 mg. per 100 ml. at 24 hours and 7.5 mg. per 100 ml. at 48 hours. In any case, the serum bilirubin should not be allowed to exceed 20 mg. per 100 ml. in the first three to five days of life.[15]

Information as to the technic and precautions to be observed in exchange transfusion is available elsewhere.[16-18] The procedure is not difficult, and with reasonable care it is relatively safe. In our last 280 consecutive exchange transfusions there has been no failure attributable to the operation itself.

Probably the best criterion for using reexchange transfusion in the moderately severe case is the serum or plasma pigment level. We have found that when pigment levels are moderately high they can be controlled by a single 500 ml. exchange transfusion for about

10 hours. A single exchange will control lower pigment levels for about 44 hours. This means that in moderately severe cases the serum bilirubin should be measured within 10 hours after the first 500 ml. exchange; in milder cases the interval can be longer. It should not, of course, be allowed to exceed 20 mg. per 100 ml. The clinician also should watch for signs of neurologic involvement. We check regularly for low-grade fever, hyperirritability, high-pitched cry, lethargy, abnormal extraocular movements, opisthotonos, dyspnea, dysphagia, heel-lifting and alteration of the Moro reflex. If the serum calcium is normal, any of these signs is indication for re-exchange transfusion. Prompt action may modify or prevent brain damage.

Detailed discussion of erythroblastosis fetalis due to such factors as Kell, Kidd and Duffy is beyond the scope of this paper. It is well to remember, however, that in many of these cases there is a maternal history of blood transfusion. The disease in the infant is similar to Rh erythroblastosis fetalis and in general requires the same treatment.

It has been gratifying to observe the recent progress in the field of the Rh and rarer blood factors, and it is pleasant to anticipate that the puzzles presented by the familiar ABO groups soon will be solved.

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Anticonvulsant Drugs

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The treatment of the convulsive disorders of childhood is a complex and difficult problem. The anticonvulsant drugs should not be considered as more than one aspect of the therapeutic approach. It is axiomatic that the attitude of the parents and the child toward the illness, factors of social and economic environment, diet, general health and emotional well-being are of paramount importance in the management of any case of childhood convulsive disorder. Further, anticonvulsant drug therapy is truly successful only when highly individualized and tailored to the particular child's needs.

Until the advent of specific anticonvulsant drugs in 1937, the clinician's approach was a simple, albeit not entirely satisfactory, one. The management of convulsions was totally dependent on two drugs, the bromide, probably first suggested by Locock in 1857, and phenobarbital, introduced by Hauptman in 1912.

With increased knowledge of the complexities of the epilepsies has come an ever-growing list of anticonvulsant drugs. From sedation, narcosis and anesthesia we have progressed to a more specific pharmacologic approach with drugs whose primary action is anticonvulsant and in which sedative and narcotic side effects are to be avoided.

It is probably impossible for any single source to produce an entirely comprehensive review of all the pharmacologic agents available for the treatment of epilepsy. Perhaps no other therapeutic problem fits so well the dictum of "many treatments, no best treatment." There are, however, certain general pharmacologic and clinical principles which can be established for our broad clinical guidance.

To discuss adequately the pharmacology of anticonvulsant drugs it is necessary first to establish a clinical outline of the epilepsies. The clinician's first mandate is to differentiate between the treatable symptomatic epilepsies due to infection, brain tumor or other brain damage, and the idiopathic forms. It should be stressed that even convulsions due to brain tumor may respond to anticonvulsant therapy for a time, but the symptomatic type of seizure usually responds less well than the idiopathic group. Drug response should not be considered a diagnostic test, except perhaps in some of the epileptic equivalent disturbances of behavior. It also should be stressed that the treatment of epilepsy by drug therapy is always symptomatic. Perhaps recent re-

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ports of the use of pyridoxine and the experimental studies on glutamine and asparagin will prove exceptions to this clinical truism.

To approach the drug therapy of convulsions on anything other than a trial-and-error basis, some correlation between symptoms, anatomy, physiology and pharmacodynamics must be attempted in every case.

Classification of Epilepsies

Recent clinical and electro-encephalographic studies necessitate reappraising the classification of the epilepsies. At present consideration of the classic clinical forms of grand mal, petit mal, focal seizures and the epileptic equivalents together with their electro-encephalographic concomitants leads to a tentative classification of seizures into two basic types: (1) those which are precipitated from a portion of the cerebral cortex and (2) those which arise from the upper brain stem, diencephalon and mesencephalon. The latter have been called "highest level seizures" and are coming to be thought of as "centrencephalic" forms.

Relating this classification to the older terminology we find that generalized convulsion manifestations may take the form of petit mal, or the more dramatic grand mal, and usually arise from trigger points in the diencephalon, mesencephalon and upper brain stem. On the other hand, partial or focal seizures usually arise from cortical trigger points. This latter form may progress from a focal onset to a major grand mal attack. It is also known that some focal attacks may result from trigger points in the infracortical "centrencephalic" system.

As a result of these considerations it now becomes necessary to analyze the seizure pattern both clinically and electro-encephalographically in order to determine the cortical or infracortical origin of the abnormal discharge. Unfortunately, such an analysis is not always possible but, if accomplished, materially aids in selecting more specific anticonvulsant drugs. As important as such an approach may be for the immediate practical problem of the patient, it is perhaps even more important in terms of the future development of more effective and highly specific anticonvulsant drugs.

It has been known for some time that certain drugs are most effective in certain types of seizures. The foregoing brief comment is a hypothetic attempt to place these empiric observations on a more systematic basis. With the ever-increasing number of drugs available for treatment, some order seems helpful in the chaos of trial-and-error clinical observation.

Before discussing specific pharmacologic agents, a basic generalization regarding dosage in children is in order. There are average or conventional starting doses for all the drugs to be mentioned; however, the proper dose for the child is not finally determined by weight or age but is that dosage which is required in order to stop the seizures of the individual child and which does not cause toxic or undesirable side effects in that particular patient. It is my personal observation that most clinicians tend to undertreat rather than overtreat pediatric convulsive problems.

Pharmacologic Agents

A definitive listing of drugs used in the treatment of seizures is probably impossible. An admittedly incomplete compilation of 14 widely used drugs and 39 less widely used or experimental agents gives some idea of the numerical problem the clinician is forced to deal with.

It is possible to group the more widely used anticonvulsant drugs chemically. There are four main groups—(1) barbiturates, (2) hydantoins, (3) oxazolidinediones and (4) succinimides. As will be pointed out later, there are other chemical types in use or under investigation which do not readily fall into those major groupings.

If we consider the cortical group of seizures, regardless of basic etiology, it becomes apparent that the hydantoin and barbiturate groups of drugs are most reliable. DILANTIN® (5,5-diphenylhydantoin sodium) and phenobarbital (5-ethyl-5-phenylbarbituric acid) are the drugs of choice in the grand mal group of either the idiopathic or secondary symptomatic type. MEBARAL® (*N*-methylethylphenylbarbituric acid) or GEMONIL® (5,5-dimethyl-l-

TABLE 1

ANTICONVULSANT DRUGS

PRODUCT	CHEMICAL COMPOSITION	MANUFACTURER
Benadryl	2-(benzhydryloxy)-*N,N*-dimethylethylamine hydrochloride	Parke, Davis & Company Detroit, Michigan
Benzedrine	dl-α-methylphenethylamine	Smith, Kline & French Laboratories Philadelphia, Pennsylvania
Dexedrine	d-α-methylphenethylamine sulfate	Smith, Kline & French Laboratories
Diamox	acetazoleamide	Lederle Laboratories Division American Cyanamid Company Pearl River, New York
Dilantin	5,5-diphenylhydantoin sodium	Parke, Davis & Company
Dimedione	3-ethyl-5,5-dimethyl-2,4-diketo-oxazolidine	Leo Pharmaceutical Products Copenhagen, Denmark
Gemonil	5,5-dimethyl-l-methylbarbituric acid	Abbott Laboratories North Chicago, Illinois
Hibicon	N-benzyl-B-chloropropionamide	Lederle Laboratories Division American Cyanamid Company
Mebaral	*N*-methylethylphenylbarbituric acid	Winthrop-Stearns Inc. New York, New York
Mesantoin	3-methyl-5,5-phenylethylhydantoin	Sandoz Pharmaceuticals Hanover, New Jersey
Milontin	N-methyl-a-phenyl succinimide	Parke, Davis & Company
Miltown	2 methyl-2-N-propyl-1,3 propanediol	Wallace Laboratories, Inc. New Brunswick, New Jersey
Mysoline	5-phenyl-5-ethyl hexahydropyrimidine-4, 6-dione	Ayerst Laboratories New York, New York
Nuvarone	3-methyl-5-phenylhydantoin	Abbott Laboratories
Paradione (Paramethadione)	3,5-dimethyl-5-ethyloxazolidine-2,4-dione	Abbott Laboratories
Phenobarbital	5-ethyl-5-phenylbarbituric acid	
Phenurone (Phenacemide)	phenacetylurea	Abbott Laboratories
Prenderol	2,2-diethyl-1,3-propanediol	E. R. Squibb & Sons Division of Mathieson Chemical Corporation New York, New York
Thiantoin	sodium 5-phenyl-5(2-thienyl) hydantoinate	Abbott Laboratories
Tolserol	3-ortho-toloxy-1,2-propanediol	E. R. Squibb & Sons Division of Mathieson Chemical Corporation
Tridione (Trimethadione)	3,5,5-trimethyl-2,4-oxazolidinedione	Abbott Laboratories

methylbarbituric acid) may be successfully substituted for phenobarbital. MESANTOIN® (3-methyl-5,5-phenylethylhydantoin), a relatively toxic drug of the hydantoin family, or PHENURONE® (phenacetylurea), may be resorted to if careful watch is kept for hematologic and hepatic signs of toxicity. MYSOLINE® (5-phenyl-5-ethyl hexahydropyrimidine-4,6-dione) and HIBICON® (N-benzyl-B-chloropropionamide) are newer drugs said to be helpful in the grand mal form of epilepsy. These latter are not as effective but certainly may be indicated where the more usual drugs prove unsatisfactory.

Viewed in order of value and safety, Dilantin leads the list, followed by phenobarbital (or Mebaral), then Mesantoin, Mysoline and finally Phenurone. NUVARONE® and THIANTOIN® (both hydantoin drugs) are successful but are considered too toxic for general use. There are many who would include Mesantoin and Phenurone in this latter category. The bromides, while effective, are less so than Dilantin and/or phenobarbital and are also more toxic. Few clinicians use bromides except in special circumstances.

Before discussing the drugs commonly used for the subcortical forms of epilepsy, the dosage, method of administration and toxicity of the already noted drugs should be outlined. Phenobarbital, available in elixir, tablet or enteric-coated form and for injection, can be given in doses ranging from 8 mg. (⅛ gr.) to 100 mg. (1½ gr.). The usual dosage in children is 16 to 32 mg., two to three times a day. Toxic effects, aside from drowsiness and occasional increased irritability, are rare and involve the skin. Rare idiosyncratic toxicities have been reported. Mebaral and Gemonil have similar toxic effects but cause less drowsiness. Excitement is not as frequent as with phenobarbital. The dosage of Mebaral is roughly twice that of phenobarbital. This drug is dispensed in 32, 50, 100 and 200 mg. tablets. Gemonil is available in 100 mg. tablets and the dose is 50 to 100 mg., two to three times a day in children. This is less effective than phenobarbital or Mebaral in most cases.

The hydantoin drugs, the basis of control of major convulsive seizures of cortical origin, are available as Dilantin or Mesantoin. Other hydantoins are either too toxic or ineffective. Dilantin is marketed in a wide range of forms and dosages each of which have their place in meeting the special needs of individual patients. There is an oral liquid and an injectable preparation. There are capsules of 32 and 100 mg.; 100 mg. delayed-action capsules; capsules containing 100 mg. of Dilantin, 30 mg. of phenobarbital and 2.5 mg. of desoxyephedrine hydrochloride; and a capsule combining 100 mg. of Dilantin and 16 mg. of phenobarbital, as well as a capsule combining 60 mg. of Dilantin with 90 mg. of Mebaral. A gelatin capsule containing 100 mg. of Dilantin in a vegetable oil is available to allay the occasional gastric irritation seen with other forms of administration. For the benefit of the infant a 50 mg. INFATAB® is marketed. This can be chewed or left to dissolve in the mouth. Dosage of Dilantin ranges from 32 to 100 mg. two to four times a day depending on the needs and the drug tolerance of the individual patient. Younger children are usually best treated with the Infatab or oral liquid form.

The toxic effects of Dilantin include ataxia, nystagmus, diplopia, hyperplasia of the gums and hirsutism. Gastric irritation and headache are infrequent. Although bone marrow depression has been reported, there is some doubt as to whether Dilantin was the responsible agent, and in any event hemopoietic effects are so rare as to be nonexistent. Skin rashes are frequent. These may occur within the first 10 to 14 days of therapy and are probably sensitivity reactions. Other toxic skin manifestations can occur and on rare occasions are serious or fatal. Despite this impressive array of occasional toxic manifestations, Dilantin is the most effective, least toxic and most widely useful anticonvulsant drug available for the management of major grand mal or focal cortical seizures in either child or adult regardless of the basic pathogenesis of the seizures.

Mesantoin, a severe bone marrow depressant, is an effective agent in the cortical grand mal and focal epilepsies. Considerable caution needs to be exercised in using this drug and it should never be selected as the first hydantoin to be tried. Mesantoin is available in 100 mg. tablets and as HYDANTAL®, 100 mg. of Mesantoin with 16 mg. of phenobarbital. The average beginning dosage for children is 50 to 100 mg. two to three times a day.

The pharmacologic management of petit mal, myoclonic and other highest level or centrencephalic seizures is, at the present time, oriented around the oxazolidinedione drugs. TRIDIONE® and PARADIONE® are most widely used and useful. Tridione (3,5,5-trimethyl-2,4-oxazolidinedione) is a relatively toxic drug causing bone marrow depression, skin rashes and photophobia. As a consequence,

Paradione (3,5-dimethyl-5-ethyloxazolidine-2, 4-dione), a less effective but far less toxic analogue, is usually the starting drug of choice. Both of these drugs are available in solution and in 300 mg. capsules. Dosage ranges from 150 to 300 mg. two to four times a day.

MILONTIN® (N-methyl-a-phenyl succinimide) is a newer compound and a virtually nontoxic drug. It has been in general use only a short time and its effectiveness is not completely established; opinions vary as to its efficacy in petit mal. This compound is available in 500 mg. capsules and is given in doses ranging from two to six capsules a day.

Many children with petit mal epilepsy develop the grand mal form of seizure, and thus it is good general practice to use both Dilantin and/or phenobarbital in addition to the more specific petit mal drugs, Tridione, Paradione and Milontin.

Psychomotor epilepsy, one of the most difficult diagnostic and therapeutic problems, should always be suspected in any case of atypical minor or petit mal convulsion. Its differentiation from petit mal is materially aided by the electro-encephalogram. Furthermore, the electro-encephalogram will help in determining the cortical, temporal lobe, or subcortical, centrencephalic origin of the attack. In the former the hydantoin and barbiturate drugs are suggested. Phenurone, which is phenacetylurea, available in 300 and 500 mg. capsules may be resorted to, but this drug is a serious bone marrow and liver toxic agent. If used, Phenurone requires monthly blood and liver studies to prevent the development of irreversible damage. Where lower level origin is suspected, Tridione and its analogue, Paradione, may be helpful.

These are the most widely accepted pharmacologic agents for treating the principal convulsive disorders. Many other drugs are in use in the treatment of convulsions in children. Little is to be gained by a simple listing. However, some general grouping of these agents is possible although admittedly theoretic and subject to considerable revision and criticism. Such a grouping is of some practical interest in treatment as well as in selection of new drugs for clinical trial.

Glutamic acid, pyridoxine, asparagin and glutamine are all apparently essential metabolic factors. Experimentally and in the occasional clinical case they may be of adjuvant value.

DIMEDIONE®, a new oxazolidinedione compound, may prove of value in the subcortical petit mal group of seizures.

MYANESIN®, PRENDEROL® and MILTOWN®, together with Rauwolfia and chlorpromazine, have certain theoretic and experimental value in the centrencephalic groups of seizures. Clinically these drugs are either impractical or incompletely investigated. They probably act by inhibiting excessive spread of impulses in the subcortical gray masses.

BENZEDRINE®, DEXEDRINE® and BENADRYL® are also drugs which act at subcortical levels and are of occasional value in the petit mal type of seizure. Some workers report excellent results with the amphetamine drugs but the diones remain the first choice of most clinicians in treatment of petit mal epilepsy.

DIAMOX®, a sulfonamide derivative and a carbonic anhydrase, is believed to have a therapeutic effect in the control of major seizures by its ability to retard the complete recovery of nerve reactivity. Its use is limited but is perhaps worth considering as an adjuvant to more established pharmacologic agents.

Summary

Despite the relatively large number of pharmacologic compounds available, it is possible to make a few basic generalizations concerning the anticonvulsant drugs:

1. In major seizures and partial or focal seizures of cortical origin the barbiturates and hydantoin drugs are most valuable.

2. The oxazolidinediones are the drugs of choice in the subcortical petit mal seizures.

3. Other types of compounds may be of value depending on site of action and the individual requirements of the patient.

4. Therapeutic efficacy must always be weighed against toxicity and less dangerous but equally sedative or narcotic effect.

5. Each child has a highly individual drug and dosage requirement.

Trauma in Rural Areas

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ACCIDENTS account for 24 per cent of all deaths in the United States of persons less than 40 years of age. Of those who live to be 40 years and over, only 5 per cent die accidental deaths.

In Kansas in 1950 and 1951 almost two-thirds of the accidental deaths were of males (figure 1). Only in home accidents did the female accidental death rate exceed that of the male. During this two year period, males accounted for almost three-fourths of the motor vehicle accidental deaths and for an even larger percentage of public accidental deaths. Females accounted for only a minor percentage of occupational accidental deaths.

Farming is a particularly hazardous occupation. Of 15,000 workers killed on the job in the United States in 1952, approximately 3800 were killed in farm work, more than in any other major industry. Figure 2, taken from the *Farm Safety Review*, a publication of the Farm Division of the National Safety Council, shows, however, that the accidental death rate in farming was exceeded by rates in extractive and construction industries. Table 1, taken from the same publication, gives the total of farm resident accidents in the United States in 1952. There were 14,000 deaths, a rate of 62.2 per 100,000 farm residents, and 1,200,000 injuries. Occupational deaths were second to motor vehicle deaths among farmers, and the occupational injuries were second to the number of injuries that occurred about the farm home. The latter include accidents during the performance of ordinary household duties or light farm duties, commonly designated as chores.

The accidental deaths in Kansas during

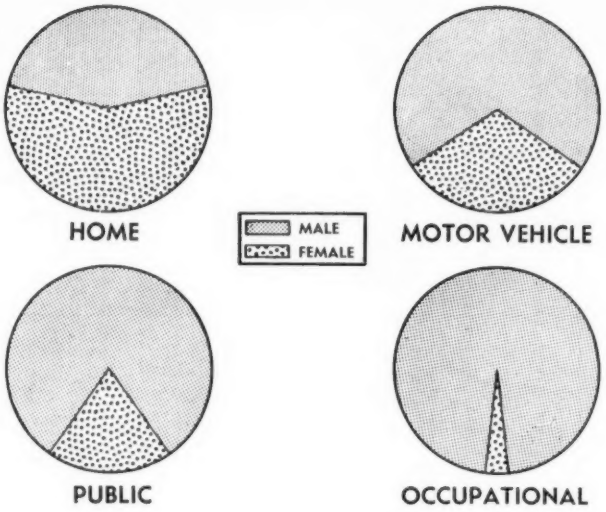

FIGURE 1. Sex distribution of accidental deaths within major classifications occurring in Kansas during the two year period 1950 and 1951.

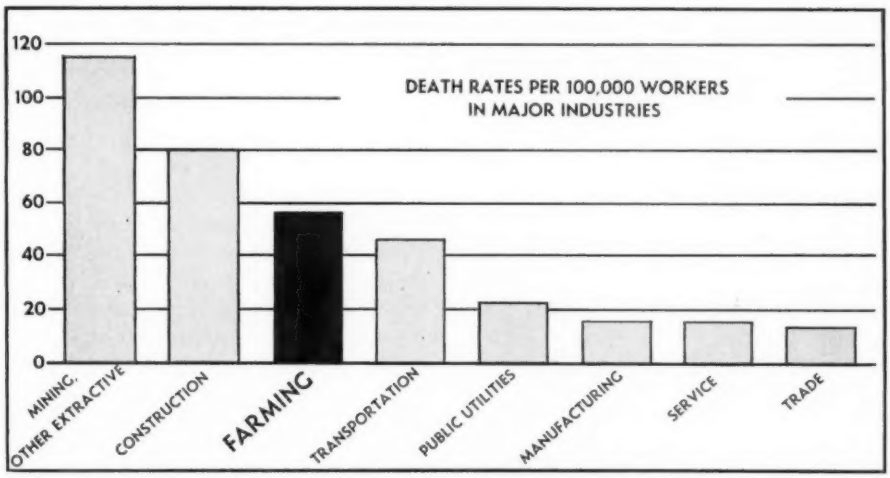

FIGURE 2. Accidental death rates in major industries, demonstrating that the hazards of farming are exceeded by those of extractive and construction industries.

TABLE 1

FARM RESIDENT ACCIDENTS, 1952

TYPE	DEATHS	DEATHS PER 100,000 FARM RESIDENTS	INJURIES
Motor vehicle	6000	26.7	210,000
Home	3500	15.5	540,000
Occupational	3800	16.9	320,000
Public non-motor vehicle	1300	5.8	150,000
TOTALS	14,600	64.9	1,220,000

FIGURE 3. Percentage of total accidental deaths occurring within each of the major classifications in Kansas during 1950 and 1951.

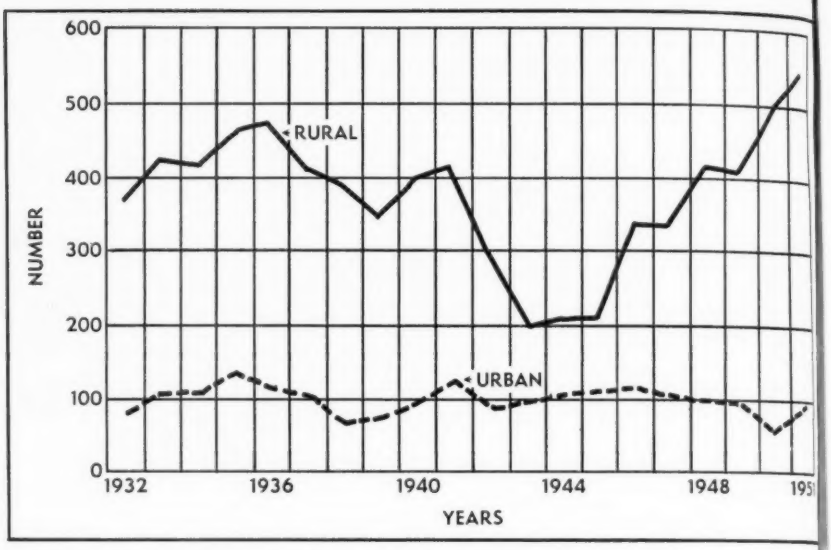

FIGURE 4. Chart depicting the number of motor vehicle fatalities in rural and in urban areas, based on 20 year survey (1932 to 1951, inclusive) by the Kansas State Board of Health.

1950 and 1951 were the result of motor vehicle accidents in 42 per cent of cases; home accidents in 33 per cent; public accidents, exclusive of motor vehicle, in 13 per cent, and occupational accidents in 10 per cent (figure 3). The farmer, of course, accounted for his share of the motor vehicle accidents. Figure 4 portrays the motor vehicle fatalities as related to rural and urban areas. Motor vehicle fatalities occurred far more frequently in rural than in urban areas. During the war years rural motor vehicle fatalities were greatly reduced in number.

Deaths due to occupational accidents in Kansas in 1950 are classified in figure 5. Agriculture accounted for 37 per cent of these fatalities.

The Kansas State Board of Health, recognizing the importance of accidental deaths as a public health problem, through its Division of Vital Statistics has accumulated and published much fine data on the subject. A report on agricultural accident mortality in Kansas made in 1950 covered the preceding 20 year period. During this time 567 persons died from accidents involving agricultural machinery, and in 307 of these deaths the tractor was the injuring agent. Of the 318 persons who died as a result of injury by animals, 130 were kicked by animals, 72 were thrown or fell from horses, 63 were gored or crushed by bulls, and 53 were injured by horses, mules, cattle or other animals. During the same period 189 persons died from falls, 163 from ve-

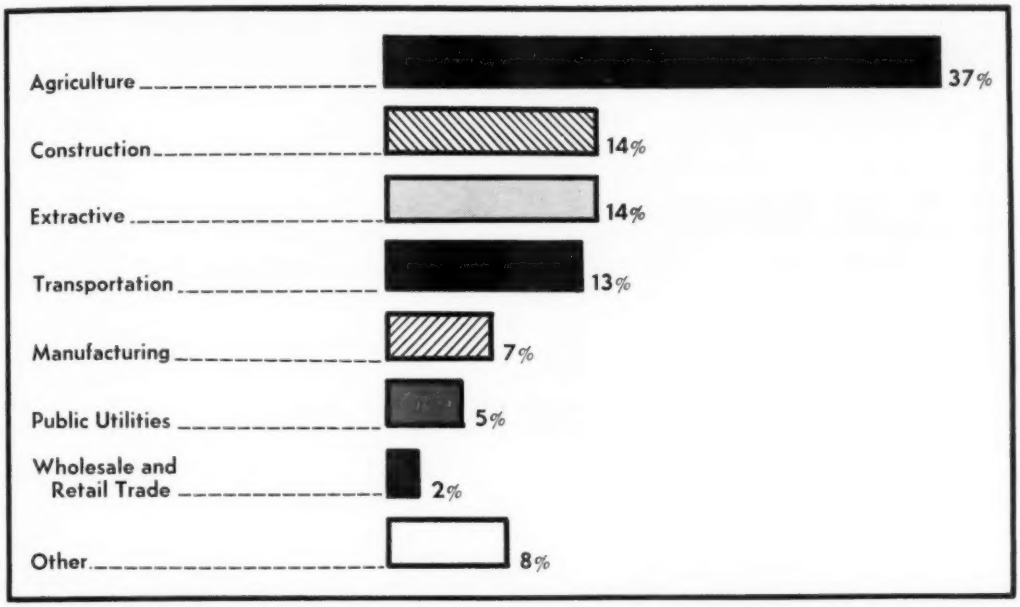

FIGURE 5. Distribution of deaths due to occupational accidents among various types of industry. (Kansas, 1950.)

hicular accidents on the farm, 114 from excessive heat, and 102 from lightning. Burns and injuries resulting from falling trees followed in the line of frequency. Other causes of death included drowning, injury by firearms, absorption of poisonous gas, dynamite explosion, plane crashes while spraying wheat fields, and poisoning by venomous animals.

Figure 6 shows the major causes of fatal farm work accidents. In Kansas during the period of 1930 to 1951 agricultural machinery accounted for 33 per cent, and injury by animals 18 per cent. Falls, vehicular accidents, excessive heat, lightning, burns and other causes accounted for the remainder.

With their increasing use, the number of fatal accidents involving tractors has increased. In 1951, 56 per cent of fatal accidents on the farm were caused by agricultural machinery, and 49 per cent were due to the tractor (figure 7). Other machinery involved in fatal accidents in 1950 and 1951 included the combine, the hay rake, plow, harrow, cornstalk cutter, corn picker, corn press and feed grinder.

The next statistics relate to admissions to the surgical service of the Snyder Clinic in St. Mary's and Wm. Newton Memorial hospitals in Winfield, Kansas. Winfield, a town of 10,000 people, is located in the Walnut River Valley and near the Arkansas River Valley where wheat as well as oats, corn, sorghum grains and alfalfa are grown. Some 10 to 15 miles east of Winfield lie the blue stem pastures of the Flint Hills in which the cattle industry predominates. Oil fields are scattered throughout all of this territory. There are a fair number of small manufacturing industries, grain elevators and flour mills.

During 1951 and 1952, 289 individuals with accidental injuries were admitted to the surgical service in the two hospitals. A classification of these accidental injuries is seen in figure 8. Home injuries constituted 31 per cent of the admissions; motor vehicle injuries 27 per cent; public injuries, exclusive of motor vehicle, 22 per cent, and occupational injuries 20 per cent. Of the 59 admissions due to occupational injuries, 24 were related to the profession of agriculture. The oil fields ac-

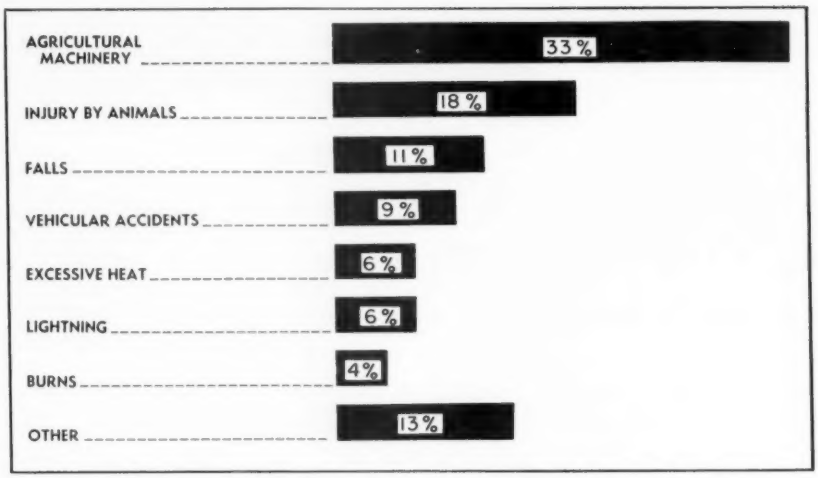

FIGURE 6. Causes of accidental farm fatalities in Kansas, 1930-1951, inclusive.

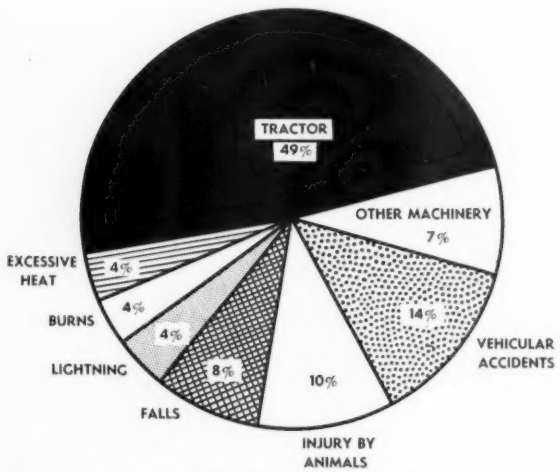

FIGURE 7. Causes of accidental farm fatalities in Kansas for the year 1951.

counted for 15 admissions, construction six, public utilities three, transportation three, and others eight.

Of the 24 admissions due to occupational farm injuries, farm machinery accounted for 12, injury by animals seven, vehicles two, and others three. Of the accidental injuries due to farm machinery the tractor accounted for 7 of the 12, two were associated with the combine, and one each with the corn picker, ensilage cutter and a circular saw. Of the seven

FIGURE 8. Percentage of accidental injuries within each of the major classifications, based on hospital admissions (surgical service) in 1951 and 1952.

injuries due to animals, horses accounted for four, and a calf, a boar and pigs the other three. The large cattle ranches east of Winfield perhaps account for more than the usual incidence of injuries due to animals. In addition to those listed as occupational, two of the injuries classified as home injuries and one of those listed as public injuries occurred when individuals fell or were thrown from a horse.

The farmer is particularly liable to accidental injury from machinery. Mechanized equipment has replaced the horse and most of the farm laborers. With the tractor the farmer may plow, disk, cultivate, mow, rake, stack, combine his wheat, dig holes for fence posts, drive fence posts, grade roads, excavate ponds, terrace his fields, move heavy equipment and perform many other tasks quickly and efficiently. He must be familiar with many types of machinery as well as gasoline engines and electric motors. He frequently repairs and maintains his own equipment.

With the advent of mechanization on the farm came a wave of accidental injuries to the farmer. Kansas was among the first states to recognize the need for a safety program. In 1938 the Kansas Farm Accident Prevention Committee published a booklet, "Safety First in Kansas Farming," which was reprinted and circulated not only in Kansas but throughout the United States. The first definite farm safety education began in about 1934 when the Agricultural Extension Service, in two or three states, announced farm safety activities through its 4-H Clubs. Minnesota was first in this safety program, followed shortly by Ohio, Kansas and Illinois, all working independently.[1] This program has mushroomed so that now 400,000 teen-agers from 46 states are enrolled in the National 4-H Farm and Home Safety Program.

The National Safety Council, through its Farm Safety Committee, later designated as the Farm Safety Conference, has been most active in this educational program. In 1947 it organized a National Farm Safety Week.

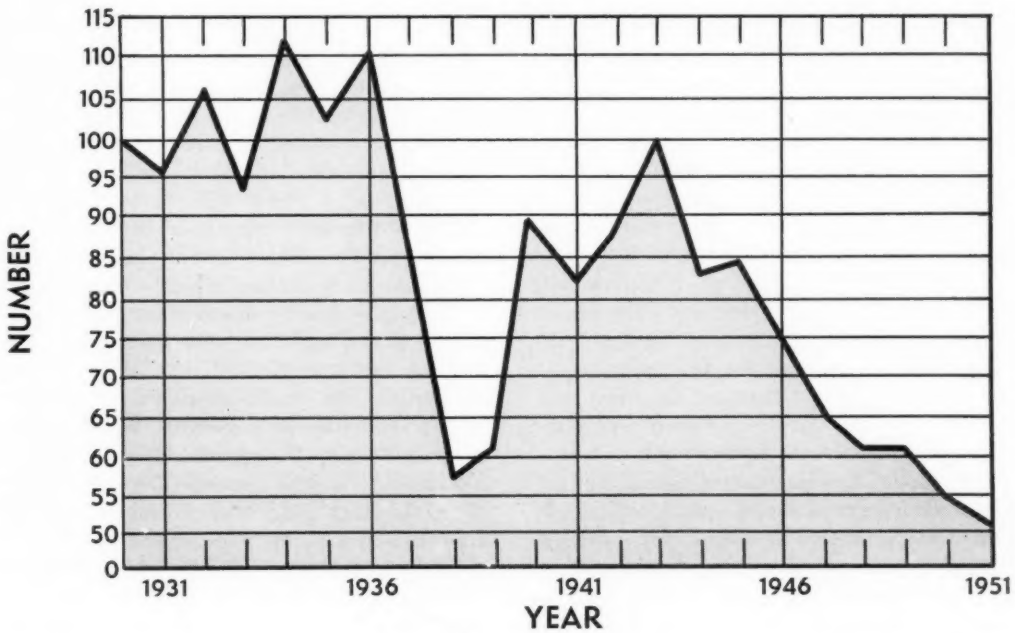

FIGURE 9. Decrease in number of accidental deaths occurring in agriculture in Kansas over a 20 year period (1930-1951). (Figures 1, 3, 4, 5, 6, 7 and 9, courtesy Kansas State Board of Health.)

The United States Department of Agriculture, the Future Farmers of America, some of the large implement companies, all the farm organizations, the Agricultural Extension Service, vocational agricultural departments in schools, the cooperatives, civic groups, the oil companies and a large milling company have entered into this safety program. Special recognition is due the farm equipment industry for its contribution. Many pamphlets have been prepared and distributed to those who live on farms. In addition to such pamphlets there have been many meetings for farm organizations where farm safety movies and other visual aids have been shown and safety speeches made.

Dr. J. H. Powers[2] wrote on the hazards of farming in the *Journal of the American Medical Association* in 1939. He and R. A. Calandruccio also reported on farm accidents to the American Association for the Surgery of Trauma in 1949.[3] Their report concerned persons injured in farm accidents in the neighborhood of Cooperstown, New York, and treated in a general hospital there. O. S. Randall of Watertown, South Dakota, in discussing this paper, called attention to the greater incidence of injuries due to mechanized equipment in that part of the country, with particular reference to the farm tractor. The fatalities and injuries reported from Kansas show a much larger percentage due to the farm tractor and other farm machinery than was the case in the patients admitted to the Cooperstown hospital.

The Kansas State Board of Health has long recognized the problem and not only has reported the accident mortality but also has made effective suggestions for reducing the accident and fatality rates on the farm. Figure 9 shows that this comprehensive program has led to a decrease in agricultural deaths. Improvement was interrupted during the war years when farm production increased rapidly and safety education activities were curtailed. An all-time low in fatal farm accidents was achieved in 1951, when there were only 51 deaths. This was 50 per cent below the record high of 112 farm accident deaths in 1934.

The management of wounds and injuries received in farm accidents is quite comparable to the management of those incurred in war. Evacuating the injured individual to the hospital is not nearly as difficult today as in former years when there were fewer good roads, ambulances and automobiles.

Tetanus immunization will frequently be indicated. When active immunization with tetanus toxoid has never been accomplished or when more than four years have elapsed since the last dose of tetanus toxoid, passive immunization should be secured with tetanus antitoxin. These individuals should also receive their first dose of tetanus toxoid, which should be administered in twice the usual dose. The second and third immunizing doses should be given in four to six weeks and 6 to 12 months.

If the last dose of tetanus toxoid was given within the preceding four years, the usual 0.5 cc. booster dose of fluid tetanus toxoid is all that is necessary. A recent report to the American Association for the Surgery of Trauma[4] summarizes current thought on this subject.

The incidence of contamination of wounds with clostridial organisms is probably higher in farm injuries than in injuries elsewhere in civilian life. Wounds produced by crushing, mangling and contusing agents on the farm provide a favorable medium for the growth of clostridial organisms and the development of anaerobic cellulitis and clostridial myositis or gas gangrene.

The differential diagnosis of clostridial myositis and anaerobic cellulitis may be most important in managing wounds incurred in agricultural pursuits. MacLennan[5] found that a large percentage of wounds incurred on the field of battle in North Africa showed contamination with clostridial organisms, yet anaerobic cellulitis or clostridial myositis developed in only a few cases.

Perhaps it is not common knowledge that clostridial organisms may invade dead and devitalized tissue, thus leading to the development of anaerobic cellulitis which in itself does not endanger life or limb. Surgery in these cases discloses the involvement only of tissue devitalized by the injuring agent and of fascia extending beyond the immediate

area of the wound. Involvement of live, healthy muscle away from the contused area is not found. The patients usually have little fever or constitutional reaction of any sort and respond well to local excision of devitalized tissues and wide incision of areas proximal to the wound to permit excision of involved fascia and the ingress of air to these parts.

When clostridial myositis or true gas gangrene develops, muscle beyond the area of traumatization will be involved and the patient will be extremely toxic and ill. Rarely, excision of involved muscle groups may affect a cure if large doses of penicillin are given. In most instances, however, amputation is necessary to save life. In many cases the diagnosis can be made only at the operating table.

The surgeon who cares for persons injured in farm accidents must be able to distinguish between anaerobic cellulitis and clostridial myositis at the operating table. Knowledge that the presence of clostridial organisms, gas, a disagreeable odor, pockets of gas in soft tissue as evidenced by radiographs of an extremity, and necrotic, foul-smelling tissue in the wound do not necessarily mean gas gangrene may save a limb for the individual who has anaerobic cellulitis. The ability to recognize involvement of muscle by an infection caused by clostridial organisms, together with the knowledge that radical surgical measures, usually amputation, will be necessary, may save the lives of individuals with true clostridial myositis or gas gangrene. A recent report[6] outlines more fully the principles of diagnosis and treatment and refers to excellent original articles on the subject.

Strict adherence to the principle of opening wounds widely and excising devitalized, dead and contaminated tissues, as well as removing all foreign material, is most important in wounds incurred on the farm. Delayed suture of such wounds should certainly be the rule. At the same time one must avoid needless sacrifice of tissue. In most wounds it is necessary only to excise the very narrowest of skin margins, and particularly in wounds of the hand every effort should be made to avoid unnecessary sacrifice of any part. The hand with its good blood supply is capable of remarkable recovery, and wide débridement or excision of tissue is unnecessary. Robinson,[7] in a paper recently presented before the American Association for the Surgery of Trauma, called attention to the fact that amputations have been performed needlessly for hand injuries. Even part of the hand is better than none or any prosthesis. With the remarkable ability of the surgeon to remake hands, every possible bit of living tissue should be saved.

Recent booklets prepared and distributed by the American College of Surgeons on the "Early Care of Acute Soft Tissue Injuries" and the "Treatment of Fractures" should be a part of the library of every physician who is called on to treat patients with traumatic injuries. Hampton's "Wounds of the Extremities in Military Surgery"[8] is a most excellent text on this subject, and the principles outlined therein are applicable to the management of wounds of the extremities incurred in civilian life, particularly on farms or in rural areas.

The ever-increasing mechanization of our age and the threat of atomic warfare certainly make it mandatory that all surgeons become familiar with the improvements in the management of wounds which have evolved from World War II, the Korean war and recent civilian experiences. Priorities for surgical care of patients with different types of wounds and injuries and of different wounds in individual patients should be known and recognized by all of us who may be called to serve in time of disaster.

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GENERAL PRACTICE REVIEW

Lesions of the Vulva

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THE vulva is unique in the genital canal, since only this region differs in embryologic origin from that of the internal genitalia. Although there is some difference of opinion as to the origin of the vagina, it seems logical to assume that the hymen or its residua is the approximate point of transition between the tissues of mesodermal and ectodermal origin. Lesions of the external genitalia are similar, then, to those of the skin rather than to those of other organs in the genital canal.

Inflammatory lesions are similar to those recognized elsewhere on the dermis, and are too numerous to discuss in detail. Intertrigo is one of the most troublesome and common of the lesions affecting the vulva. When accompanied by hyperhidrosis and seborrhea, particularly in the obese patient, intertrigo can be one of the most annoying benign afflictions of the human female. Attention to cleanliness and the use of drying powders are helpful in combating intertrigo. Folliculitis is a not infrequent offender as a result of local irritations. Again, local cleanliness may prevent more extensive infection. If furuncles occur, parenteral use of antibiotics may be necessary to combat deep-seated involvement.

Dermatitis as a result of sensitization to external irritants is probably more common than is suspected. Douche solutions, particularly those containing harsh chemicals not compounded for such use, local anesthetic ointments and certain soaps, especially the perfumed variety, are included in this group of sensitizing products. New girdles or other varieties of underclothing as well as pads worn at the menses are among the clothing items that may instigate pruritus.

Vulvitis is not uncommonly the result of vaginal infections, particularly those caused by fungi and *Trichomonas*. Even the discharge from nonspecific vaginitis, especially the postmenopausal type, may give symptoms referable to the vulva. It is therefore important to examine carefully and treat such con-

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ditions in order to relieve the vulval symptoms.

Although less common today, venereal diseases and allied conditions which affect the external genitalia deserve special mention in view of the changes in therapy of the acute lesion and in view also of the malignant potentialities of the chronic stages. Syphilis may be seen as primary, secondary or tertiary lesions in this region. The chancre is noted less commonly by the female than the male since it produces less discomfort both physically and visually. However, the secondary lesions, in the form of condylomata lata, are most troublesome. They not infrequently become confluent, secondarily infected and ulcerated, with resultant extensive lymphadenitis. Therapy for these lesions has been well documented in treatises on syphilis.

Lymphopathia venereum, or lymphogranuloma venereum, although relatively uncommon, is often quite insignificant in the initial phase, and as a consequence the patient seeks no aid. The sequelae take the form of late destruction of tissue with scarring and fibrosis and/or lymphedema due to obstruction of the lymphatic drainage. Rectal and urethral involvement may lead to either stricture formation or incontinence. Elephantiasis of the vulva is most frequently the end result of lymphogranuloma. Diagnosis of this virus infection often cannot be made with absolute certainty. The use of the Frei antigen intradermally will aid in the diagnosis; however, the antibodies remain for many years after the initial lesion subsides. Consequently, the presence of a positive Frei test simply indicates that the patient has at one time been exposed to the virus. The broad spectrum antibiotics, such as AUREOMYCIN®, TERRAMYCIN® and ACHROMYCIN® are felt to be of some value in treating the initial infection.

Granuloma inguinale, diagnosed when the characteristic cellular inclusions (the so-called Donovan bodies) occur in the smear or biopsy of the tissue, may be heralded by a small and inconspicuous ulcer. The spread of this lesion occasionally resembles cancer both grossly and systemically. The local lesion is exuberant, fungating and friable, and causes general systemic debility and anemia. Extragenital manifestations occasionally seen are destructive in nature. Prior to the advent of streptomycin, tartar emetic and FUADIN® were the only therapeutic agents of value; however, these were quite limited in their action and inadequate therapy for an extensive lesion. Attempts at wide local excision resulted in extensive procedures and large denuded areas which made grafting necessary. In spite of these heroic efforts, there was usually local recurrence. Streptomycin has changed this story dramatically. Reports indicate that 100 per cent cure rate can be obtained by the vigorous use of this antibiotic and others of a similar nature during the initial stages of the disease. It must be emphasized, however, that like syphilis, if the disease is not treated in the initial active phase, the destructive end results are little affected by the medication. Furthermore, both granuloma and lymphogranuloma may be precursors of carcinoma, and the gross picture of the end stages of these diseases is in no way different from the earlier stages of carcinoma. Consequently, there is often a tendency to omit the use of the biopsy in the chronic granulomatous group because of the absence of a noticeable change in the macroscopic appearance. In a recent survey of 50 cases of carcinoma of the vulva at The Johns Hopkins Hospital, 10 were preceded by such lesions, and in many the presence of the local disease had been noted for 10 years or longer.

Chancroid, another member of the venereal disease family, is initially a nonspecific ulcer occurring in the vulval region. Whereas other ulcerative lesions are commonly more or less painless, the "soft chancre" is characteristically painful in a majority of the cases. The infection, believed to be due to the bacillus *Hemophilus ducreyi*, often produces multiple small lesions which become secondarily infected, giving rise to marked inguinal adenitis. Cleanliness and the use of sulfonamides have been the therapy of choice. So far the wide spectrum antibiotics have not been used widely in view of the good results obtained with sulfonamides. Local distortion of vulval architecture is the only long-term sequela recognized at present.

Gonorrhea affects the vulva only secondarily as the structure in the adjacent area is primarily involved. In the prepuberal phase of life the vaginal mucosa is characteristically thin and susceptible to invasion by the gonococcus. The resultant purulent vaginitis produces marked vulval erythema, leading to use of the term "gonococcal vulvovaginitis." It was recognized in the work of Lewis in 1933 that local estrogen therapy to the vagina which results in proliferation of the vaginal mucosa could effectively combat the vulval condition without any additional local therapy. Similarly in adults, acute frequent edema and erythema of the vulva due to the gonococcus is secondary to infection and abscess formation in Bartholin's gland. Urethritis and Skene's gland infection also may produce adjacent reaction. Again the vulval manifestations are treated satisfactorily by therapy to the area of primary infection. Penicillin is the treatment of choice, and incision and drainage of abscess of Bartholin's gland are indicated if such occurs. Injection of penicillin under local anesthesia into the gland has been suggested, but often is as much of a procedure as actual drainage, if not more so.

Recently a new cause of acute vulvitis has appeared. As a result of treatment with antibiotics—particularly the mycin group—some patients have developed marked pruritus, both anal and vulval, with occasional extreme edema and reddening. Not uncommonly, a fungous infection is found as the cause of this vulvitis, although trichomonads are occasionally the offenders. It is assumed that normal organisms in the vagina which may control such infections are eliminated by the antibiotic therapy, and the pathogenic agents are allowed to grow and produce symptoms.

Leukoplakia and Kraurosis

No discussion of vulval lesions is complete without including kraurosis and leukoplakia. Marked divergence of opinion exists as to the exact nature, pathology and malignant potentialities of these conditions. There are those, for example, who talk of the atrophic and the hypertrophic varieties of leukoplakia, and suggest that kraurosis is really the atrophic phase of leukoplakia. Likewise, the term leukokraurosis has become a familiar one, evidently describing a white lesion which is associated with shrinking of the vulval skin. Finally, the dermatologist associates this whitish, atrophic change with lichen planus sclerosus et atrophicus. Apparently there is no uniformity of opinion as to the exact designation for the many whitish, pruritic, excoriated changes which are seen so commonly on the vulva.

In an effort to simplify the problem, the term kraurosis can first be defined as an atrophic change occurring in the vulval tissues in which there is a loss of the subcutaneous tissue, so that in the most advanced stages the thinned, atrophic epidermis appears to be almost stretched over the pubic rami. There is a concomitant contraction of the vaginal outlet so that dyspareunia is a condition usually associated with pruritus. Excoriations occur on the basis of this pruritus. Microscopically the epithelium is thinned, elastic tissue is replaced by collagen, and also there is a cellular infiltration in the dermis. This then simulates very closely the picture of lichen planus sclerosus et atrophicus. Therapy in general is rather unsatisfactory. Local application of lanolin locally is sometimes helpful. Minor plastic procedures restore a more adequate outlet. Antipruritic ointments will be mentioned later.

Leukoplakia is by definition a white patch. Consequently, it could include any chronic pruritic process which has by its irritation produced certain tissue changes. Because this change is not uncommonly associated with the later development of carcinoma, leukoplakia of the vulva has come to be associated with leukoplakia of the lip and oral mucosa, although the vulval skin is certainly not a mucous surface. Consequently, the term leukoplakic vulvitis is probably preferable and would include a variety of lesions which clinically justify the use of such a term by the appearance of white elevated patches, often associated with excoriations and even superficial ulcerations. These lesions should be biopsied, because, regardless of their gross appearance, intra-epithelial cellular activity

may be present. In this group carcinoma will develop frequently if the lesion is not removed. On the other hand, without such cellular changes, the condition may be treated symptomatically; that is, by attempts to relieve the pruritus which is present in almost all cases. Such antipruritic ointments as carbonic detergents, calamine with 1 per cent phenol, anhydrous lanolin, and more recently, HYDROCORTONE® ointment are of value. Ointments with local anesthetic agents are helpful but have the disadvantage of producing an occasional sensitivity reaction which can make matters worse. If, however, such therapy does not produce the desired results, some variety of nerve block, nerve interruption or actual excision must be considered. It should be recognized that, given a chronic irritative lesion and a cancer-susceptible patient (and this biologic or chemical group must be a real one), a malignancy will result in a certain number of cases regardless of what term is used for the gross lesion. Consequently, it is necessary to treat, biopsy and follow such patients carefully if cancer is to be prevented, or at least treated in its earliest stages.

Cystic Neoplasms

Cystic lesions of the vulva are almost never new growths. The Bartholin's duct cyst is usually the result of inflammatory blockage of the duct, although traumatic involvement in repairs and episiotomies is certainly possible. Sebaceous cysts are also inflammatory in origin. It is common to see numerous small cysts of this variety and an occasional large one. Both Bartholin's and sebaceous cysts rarely need treatment unless they become recurrently infected, and since their malignant potentialities are negligible, it is preferable not to employ surgery. Rarely, there appears a cystic dilatation in the labium majus at the site of the termination of the round ligament in this area. This is the so-called cyst or hydrocele of the canal of Nuck. Finally, a rare cyst at the end of the mesonephritic duct may appear at the outlet and seem to involve the vulva. This Gartner's duct cyst is really vaginal in origin and ordinarily removal is not necessary.

Benign Solid Neoplasms

Benign solid new growths on the vulva of clinical importance are relatively infrequent problems. Myoma, lipoma and fibroma and soft papilloma occasionally are seen on the vulva but do not differ from those found elsewhere.

Commonly seen on the vulva as well as the adjacent perineum, vagina and occasionally the cervix are condylomata acuminata, formerly known as "venereal warts." Although these often are associated with vaginal discharge, it is now generally accepted that they are viral in origin. The common symptom is pruritus, the usual symptom of most vulval lesions. Although these warts usually are small, they may become very extensive, especially during pregnancy when they tend to increase in size and number. During delivery, the vulval variety is rarely troublesome. However, the vaginal and cervical lesions may bleed due to increased vascularity. The treatment of choice for the small lesions is the application of 20 per cent podophyllin resin in tincture of benzoin base. Since podophyllin is very irritating, care should be exercised to localize the application of the solution to the lesion. Tincture of benzoin aids in attaining localization. If the verrucae persist, spread or become granular in appearance, they should be removed for microscopic study, remembering always that carcinoma may begin with such a lesion.

Nevi of several varieties are not uncommonly seen on the vulva, and since irritation is more likely in this area than most others, pigmented lesions should be removed and studied microscopically. Malignant tendencies may be noted in even the most benign-appearing nevus, and incomplete removal of a junction nevus often will lead to recurrence with possible malignant change. Capillary nevi and hemangiomas are not infrequently noted on the vulva. The former may be congenital and of appreciable size. Irritations from diapers and excreta often produce ulceration and bleeding, necessitating surgical treatment. The tiny hemangiomas are often multiple and rarely require therapy.

One of the most interesting of the group of

benign solid neoplasms is the hidradenoma. Arising from the sweat glands, the tumor more commonly is seen on the labia majora, although occasionally it is found on the labia minora. The lesion rarely attains a size larger than 1.5 to 2 cm. in its greatest dimension. It is brownish in color and projects over the surface of the skin. The center may be slightly indented or even ulcerated. The tumor is enucleated without difficulty, being grossly very well circumscribed. The most interesting aspect is the microscopic picture, which to the uninitiated may suggest malignancy because of marked variation in the size and shape of the glands. However, closer inspection reveals no anaplasia of the individual cells, although metaplasia may take place. Although a rare hidradenocarcinoma has been reported, the tremendous majority of these lesions are found to be benign.

Intra-epithelial Disease

The recognition of intra-epithelial stages of malignancy has become one of the most important advances in the study of cancer in any region of the body. Until a cure is discovered, the best chance for improvement in the salvage lies in earlier diagnosis, preferably prior to invasion. The results from treatment of intra-epithelial carcinoma of the cervix are well known—almost 100 per cent five year salvage has been recorded in the majority of adequately treated cases. Since cancer of the vulva is much less common than cancer of the cervix, the preinvasive stages of malignancy are not as easily recognized. Furthermore, biopsy of vulval lesions is not used frequently in cases of chronic disturbances, so there is little chance to observe these very early stages of the disease. There are, however, a great variety of gross pictures which may demonstrate microscopic intradermal anaplasia. A few of these have been described adequately by dermatologists. In 1912 Bowen reported two cases of the chronic, nonhealing skin disease which now bears his name. This red, oozing lesion usually has some superficial crusting and shows almost no infiltration. Occasional small white epithelial islands are seen. Microscopically there are intra-epithelial changes characteristic of anaplasia, but, at this stage, the disease is localized to the epithelium. Paget's disease of the nipple is another well-known entity, although its extramammary counterpart is less well documented, and there are those who doubt its existence. The few cases described in the literature are clinical examples of intra-epithelial malignancy with the invasive potential a remote but definite possibility if the lesion is undiagnosed and untreated.

Other gross lesions which may on occasion disclose epithelial anaplasia are chronic irritations with superficial excoriations and overgrowth of the epidermis with leukoplakoid changes; small areas of granularity which fail to heal; condylomatous lesions, particularly the recurrent variety, with oozing and excoriations; and the chronic, lymphedematous, fibrotic and ulcerative lesions of the granulomas. Consequently, a chronic, nonhealing and persistently irritating lesion which produces gross changes in the characteristic picture of the vulval skin deserves biopsy, if the earliest phases of malignant changes are to be recognized. Even though invasive carcinoma is rare, the relative simplicity of vulvectomy as opposed to the radical procedure involving lymph node dissection is enough to warrant more thorough investigation of these chronic lesions.

Malignancies of the vulva are also similar to those seen elsewhere on the skin; however, the survival rate is poorer than for the usual carcinoma of ectodermal origin. The reasons for this disparity are unknown, but two features stand out as definite possibilities: (1) Primary carcinoma of the vulva is generally a disease of old age and with old age frequently come increased modesty and increased tolerance to discomfort, and (2) the local irritants of clothing, urine, perspiration and seborrhea, superimposed on the trauma induced by scratching, may produce more rapid local and lymphatic extension. The best salvage rate for carcinoma of the vulva is in the neighborhood of 50 per cent.

Primary malignancy of the vulva comprises about 4 to 5 per cent of all malignancies of the genital canal. It is generally a disease of

old age, the average being 60 years, in those cases of unknown etiology or those associated with leukoplakia. The cases preceded by granulomatous lesions, however, are found in a younger age group. In a series of 13 cases (to be reported) preceded by such granulomas the average was 42 years. The gross appearance varies greatly, depending partially on the pre-existing lesion, if present. Postgranulomatous carcinomas often resemble the initial lesions, thus presenting the appearance of elephantiasis, markedly distorted and destroyed external genitalia, or a group of secondarily infected condylomas. Those malignancies, superimposed on leukoplakic vulvitis, may present simply excoriations or ulcerations in the leukoplakic areas. Other carcinomas developing without any apparent preexisting disease appear grossly as ulcers or granular cauliflower masses of varying sizes. Any area on the vulva may be affected initially; however, the regions of the labia minora and adjacent majora are the most frequent sites for the malignancies arising in the older age groups.

The marked variation in the gross appearance and the similarity between the pre-existing disease and its malignant counterpart are the most important features of the gross picture of vulval cancer. As is true of the cervix, if chronic, irritative lesions of the external genitalia are examined more closely and subjected to tissue study, more malignancies will be noted earlier. Furthermore, like cancer elsewhere, treatment in the early stages is simple and more satisfying. This is particularly true for vulval lesions, existing as they so often do in older women for whom hazards of radical surgery are greater.

Treatment of carcinoma of the vulva is surgical. Since Taussig's epoch-making survey of the problem, others have hastened to substantiate his findings. With the use of radical vulvectomy and inguinal and femoral lymphadenectomy, the salvage rate has almost doubled. Recently, even more radical procedures have been suggested and are in use in several clinics. The results will have to be appraised later, but an increase in operative procedures in older patients would seem bound to increase the primary mortality rate and to nullify much of the possible value of operation. Furthermore, for postgranulomatous lesions, wide local excision is of major importance. In spite of long-standing disease, nodes were involved in one of eight cases in which they were removed, and in six deaths autopsy revealed evidence of extension beyond the local area in only two cases.

X-ray therapy generally is used only as an adjunct to surgery in this country, or for palliation in very extensive cases or for poor-risk patients. An exception to this rule is suggested for those cases in which pathologic examination reveals a more poorly differentiated tumor, such as the basal cell variety. As in the case of skin malignancy of similar cell type, x-ray therapy offers more aid than in the usual spinal cell tumors. However, even here, surgery should be used as the primary treatment unless the aforementioned conditions exist.

At some variance to the mode of therapy suggested here is that used in Stockholm. At the Radium Hemmet, extensive electrocoagulation of the local growth is followed by radiation therapy or surgical excision to the nodes. Although no personal experience can be reported, it seems that this might be a worthwhile adjunct to therapeutic methods for the patient with extensive local disease or for whom surgery is contraindicated.

Although epidermoid carcinoma is the common type of primary malignancy of the vulva, there are several other varieties. Melanocarcinoma is seen occasionally and may develop in the clitoris. It is sometimes mistaken for a hematoma, which it grossly resembles. As usual, it is extremely malignant and the prognosis is poor. Primary adenocarcinoma, occasionally seen originating in the Bartholin's gland, is a problem to treat because of its extension toward the rectum. Primary sarcoma is an infrequent malignancy of the vulva.

Secondary malignancy may have its primary site in the cervix, urethra, rectum or, rarely, in the bladder. Choriocarcinoma of the uterus seems to have a predilection for the vagina and vulva, and the diagnosis has sometimes been made by biopsy of a metastasis in one of these areas.

CASE REPORT

Lipofibroma of the Larynx: Technic for Removal

DAVID A. DOLOWITZ AND THOMAS KEYES*

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THIS report is directed to a case of intrinsic lipofibroma of the larynx, to the difficulty in establishing a diagnosis, and to a technic which perhaps aided in forming a more perfect vocal cord following excision of the tumor.

Lipofibroma of the larynx is rarely encountered, even in the larger clinics. New and Erich[1] in 1938 found only one such case in a 30 year survey of cases of benign laryngeal tumors at the Mayo Clinic. Since the first case reported by Holt[2] in 1854, there have been several excellent reviews of this subject,[3-6] the first by Goldstein[3] in 1909, and the last by Saunders[6] in 1954.

Lipofibromas have been variously classified, sometimes according to the site of origin, and sometimes according to the area in which the tumor is found. For this reason, it is hard to be certain that all reported cases have been reviewed, and no attempt has been made to estimate the total number. All reviewers agree, however, that relatively few laryngeal lipofibromas are intrinsic. Birkett[1] noted the intrinsic type in but 5 of 47 cases.

DAVID A. DOLOWITZ

THOMAS KEYES

Case Report

A 13 month old white male infant was first admitted to the Salt Lake General Hospital on November 19, 1953. Two months prior to admission respiratory distress suddenly developed; it was acute for two days and then lessened in intensity. There was marked in-

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spiratory wheezing, and some sternal retraction which became very severe if the child cried or was made to lie flat. There was no associated fever, irritability, gastrointestinal upset or physical weakness. Respiratory difficulty was accompanied by trouble in feeding because of intermittent vomiting. There was no choking or nasal regurgitation of food. Despite the vomiting, the infant continued to eat well and remained rather well nourished for the two month period.

The patient's mother had developed a stiff neck about a week after the onset of the child's illness. Poliomyelitis was suspected despite the absence of other signs and symptoms. The family physician suspected that the child had bilateral vocal cord paralysis due to bulbar poliomyelitis. He was concerned because of the increasing degree of sternal depression. A roentgenogram of the chest at that time did not show any abnormality, but the sternum continued to sink. Accordingly, the child was admitted at the Salt Lake General Hospital for treatment, which was sponsored by the Poliomyelitis Foundation. The patient's past history and his family history were noncontributory.

Physical examination—Temperature was 98.6° F.; pulse 110, and respiration 46. There was obvious respiratory obstruction with retraction and inspiratory wheezing. The cry was of good quality. There was no cervical swelling or lymph node enlargement. The chest showed marked sternal, intercostal and subclavicular retraction which was accentuated by crying. The lungs had inspiratory wheezes, but no rales or rhonchi were heard. The heart rate, rhythm and size were normal and there were no murmurs. The abdomen was soft, without tenderness; the lower edge of the liver was palpable. The spleen was at the costal margin. Genitalia were normal. No neurologic abnormalities were detected.

Laboratory data—The volume of packed red cells and the sedimentation rate were normal. The white cell count was 19,300 with 20 polymorphonuclear cells, 78 lymphocytes and 2 monocytes. The urine examination and Kahn and tuberculin tests were negative. Nasal culture showed a few pneumococci; throat culture, a few alpha hemolytic streptococci. Fluoroscopy revealed that the esophagus and swallowing function were normal, but the roentgenologist noted a small round mass, 6 mm. in diameter, just below the vocal cords.

FIGURE 1. Roentgenogram showing tumor mass attached to the lower surface of the right vocal cord.

The day following admission, direct laryngoscopy revealed a tumor mass attached to the lower surface of the right vocal cord. The tumor occluded 70 per cent of the lumen of the trachea. X-ray examination confirmed the presence of the mass at this point (figure 1). That evening a tracheotomy was performed under local anesthesia, with the tube inserted through the fourth and fifth tracheal rings. Then, under light general anesthesia, the first tracheal ring and cricoid cartilage were separated in the midline and retracted laterally.

A solid tumor 6 or 7 mm. in diameter was found attached to the right vocal cord, posteriorly. Since this tumor felt fibrous as well as fatty, it was feared that unequal contraction of fibrous bands might destroy the precise approximation necessary for a good voice. A 1 per cent solution of procaine was therefore injected to distend the cord to about

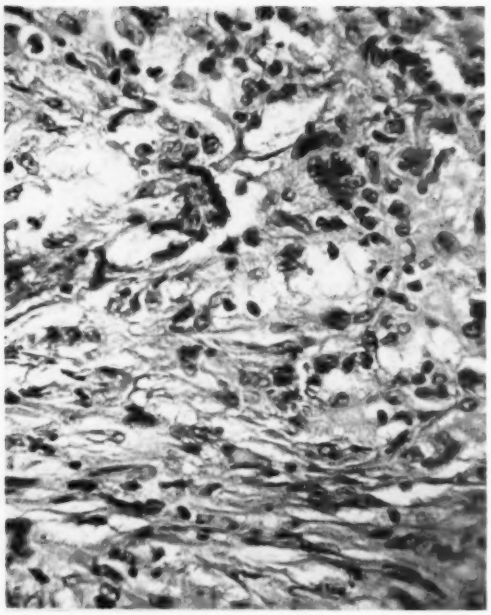

FIGURE 2. Lipofibroma of the larynx as proved by biopsy.

three times its usual size, and the mass was excised by sharp dissection. It was believed that after incision the distended tissue would contract as the solution was absorbed. It was hoped that this distention would equal the potential immediate fibrous tissue contraction and thus permit formation of a straight cord. The cricoid cartilage was approximated with stainless steel wire, and the tracheal rings were closed without suture. At laryngoscopy the next day, after the procaine solution had been absorbed, the procedure was proved successful. The cord was straight, except for slight narrowing where it attached to the arytenoid cartilage, and the voice was normal.

The excised tumor was firm and felt fat and fibrous. There was some question among the pathologists as to whether this tumor was a lipofibroma or a lipogranuloma. Studies ruled out syphilis, tuberculosis, coccidioidomycosis and other fungi. The final diagnosis was lipofibroma (figure 2).

Postoperatively, the patient's course was smooth for 24 hours; then a small area of mediastinal emphysema and slight pneumothorax at the left anterior base were observed. This air absorbed gradually and x-rays taken a week later showed a normal chest.

The tracheotomy tube was removed with no difficulty on the eighth postoperative day. The wound healed by first intention in five days. At this time the cricoid cartilage felt as firm as the thyroid cartilage. The vocal cord healed with a very slight defect close to the arytenoid cartilage but the voice was very adequate. The patient was discharged from the hospital on November 30.

When the child was seen six weeks later, he had gained 6 lb. and had grown 1 in. He was walking and running normally. A report from the family physician in April 1954 stated that the child was developing normally and that there were no signs of recurrence of respiratory difficulty or hoarseness.

Summary

Because of the similarity of respiratory distress produced by benign laryngeal tumors and by neurologic disorders, and because the symptoms appeared during a poliomyelitis epidemic, a preliminary diagnosis of poliomyelitis was made and the child was treated for this condition. This shows the necessity for laryngoscopy in all cases of respiratory difficulty.

Since a number of fibrous bands lay within the tumor approximately at right angles to the surface of the cord, simple excision of the mass would tend to leave a surface with irregular depressions due to contraction of the cut fibrous bands. By ballooning the area with submucosal injection of procaine prior to excision, the tumor could be removed with minimal irregular retraction of its base, resulting in little or no vocal cord deformity.

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CASE REPORT

Chlorpromazine-induced Jaundice

LOUIS PELNER AND SAMUEL WALDMAN

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JAUNDICE with certain special characteristics has been reported as a complication of chlorpromazine therapy.[1-7] Even though this side effect is infrequent in view of the varied and extensive employment of this drug, it is nevertheless important to know of its existence. This is true because the chemical findings in the blood and urine in cases of chlorpromazine-induced jaundice are usually those of an obstructive or regurgitative type of jaundice, which in the past has usually placed the patient in the surgical category. Indeed, many of the patients with this type of toxic jaundice have already been operated on, but no obstruction in the extrahepatic biliary tree has been found.[8] In the case which we shall report, the patient fortunately escaped this hazard, because of our awareness of this syndrome.

LOUIS PELNER

SAMUEL WALDMAN

Case Report

A 30 year old woman had suffered from "nervousness" and "irritability" for a long time. While at a country resort, she had consulted a physician, who had advised her to take one DEXAMYL® tablet, one 25 mg. tablet of THORAZINE® (chlorpromazine hydrochloride) and 10 minims of tincture of *Valeriana* three times a day. She had taken these drugs for two weeks. Nausea and vomiting had occurred one week later, and had lasted for three days. Generalized itching then had developed, and she had noted that she was passing dark urine and very light stools.

When she was first seen in the office on September 13, 1954, she was rather deeply jaundiced. There was no history of gallbladder disease or of fullness, belching or heartburn. Her diet had been poor because she had

TABLE 1

RESULTS OF BIOCHEMICAL EXAMINATION OF THE BLOOD AND URINALYSIS

DATE	BLOOD ANALYSIS						
	Blood Sugar *Mg. per 100 cc.*	Blood Urea Nitrogen *Mg. per 100*	Prothrombin Time *Seconds**	Icterus Index	Total Serum Bilirubin *Mg. per 100 cc.*	Serum Cholesterol *Mg. per 100 cc.*	Serum Cholesterol Esters *Mg. per 100 cc.*
9/15/54	122	13.4	13	41.0	7.0	437	200
9/17/54				58.4		409	208
9/20/54	122	25.0			5.8	380	230
9/24/54				43.7	5.8		
9/27/54	115	15.6	13	37.0	5.1	290	158
10/1/54				34.0	3.35		
10/4/54	102	18.0	13	23.4	2.05	310	195
10/8/54				22.0	1.15		
10/11/54	100	17.0	13	16.4	1.65	281	
10/15/54				15.0	1.5		
10/20/54				6.0	0.6		

*The normal prothrombin time was 13 seconds.

cooked most of her own meals at the resort. On week ends she had taken her dinners at a restaurant.

With the exception of the very obvious jaundice, the results of physical examination were singularly negative. The liver and spleen were not palpable, but the liver was tender to heavy percussion over the lower ribs. Office urinalysis revealed a large amount of bile, but urobilinogen was not present. The patient's general physical condition appeared to be satisfactory.

She was admitted to the Swedish Hospital on the following day (September 14). She was kept in bed during the 31 days that she remained in the hospital. During this time, she was practically afebrile.

The biochemical findings in this case (table 1), like those in many other cases in which jaundice has resulted from the administration of chlorpromazine, were indicative of obstructive jaundice, but, in cases of this type, the obstruction is intrahepatic, that is, in the small bile canaliculi. On this basis, an intravenous infusion of 5 cc. of DECHOLIN® sodium and 1000 cc. of a 5 per cent solution of dextrose was administered daily. It was reasoned that the diluted solution of Decholin sodium would cause a flushing of the small bile canaliculi.[9] Since improvement did not occur, and since the icterus index actually increased, this therapy was discontinued after four days.

During the next 16 days (from September 18 to October 3, inclusive), the therapy included the daily administration of a high potency vitamin B complex by the intramuscular route, and the use of a diet that contained a large amount of carbohydrate, a moderate amount of protein and a small amount of fat. During the same period, 20 mg. of sodium adenosine-5-monophosphate in gelatin solution (MY-B-DEN®) was administered daily by the intramuscular route because of the presence of severe pruritus. We previously had found that this preparation had a salutary effect on the pruritus of jaundice, as a temporary measure prior to operation.[10] In this case, it diminished the itching markedly, but did not eradicate it. The administration of this drug and the vitamin B complex was discon-

TABLE 1

RESULTS OF BIOCHEMICAL EXAMINATION OF THE BLOOD AND URINALYSIS

BLOOD ANALYSIS					URINALYSIS		THERAPY
Cephalin-Cholesterol Flocculation	Serum Alkaline Phosphatase *Bodansky units per 100 cc.*	Total Serum Protein *Gm. per 100 cc.*	Serum Albumin *Gm. per 100 cc.*	Serum Globulin *Gm. per 100 cc.*	Biluria, Grade	Urobilinogen, Dilution of Urine	
1+	16.8	6.6	3.56	3.04	4+	0	Decholin sodium
0	17.8	6.85	3.1	3.75	4+	1:20	
					4+	1:20	Vitamin B complex and sodium adenosine-5-monophosphate
0	18.6	8.0	3.48	4.52			
					4+	1:20	
0	14.5	6.40	3.28	3.12			Hydrocortisone**
					Trace	1:10	
0	19.2	7.15	2.90	4.25	0	1:10	Choline dihydrogen citrate
	12.0	6.2	3.4	2.80	0	1:1	

**Hydrocortisone was administered for four days, namely, from October 4 to October 7, inclusive.

tinued at the end of the 16 days (October 3).

Administration of hydrocortisone was started on the following day,[11] 10 mg. daily. Since this drug did not produce any appreciable improvement, its administration was discontinued after four days (October 7).

During the remaining seven days that the patient was in the hospital, 4 gm. of choline dihydrogen citrate was administered daily. When the patient was dismissed from the hospital on October 15, she had recovered almost completely. When the patient was examined on October 20, there was no visible evidence of jaundice, and she felt quite well. Although the icterus index and the value for the serum bilirubin were normal, the value for the serum alkaline phosphatase was still moderately elevated, and the values for the serum albumin and the serum globulin were tending to return to normal.

On October 25, cholecystography was performed by administering the contrast medium orally. It revealed that the gallbladder was functioning normally, and it did not disclose any evidence of gallstones.

Comment

Although jaundice resulting from chlorpromazine therapy appears to be infrequent, it is, nevertheless, very important that its possible presence be recognized. Any drug that is capable of causing clinical jaundice must be used with great circumspection. We have found Thorazine to be an extremely useful drug in our practice, but we feel that it must be used only for very definite and precise indications. Thus, it assuredly must not be used when other, safer, drugs would do as well. The indication for the use of Thorazine in this case was certainly not definite or clear-cut.

The problem of jaundice induced by chlorpromazine is complicated by the fact that chlorpromazine itself is apparently not a hepatotoxic drug. A study of liver function in a large series of cases in which chlorpromazine was being used did not disclose any significant abnormality.[6]

As we have mentioned previously, the results of liver function tests in the case which we have reported and in other cases in which jaundice has resulted from the administration

of chlorpromazine were similar to those obtained in cases of obstructive jaundice. The values for the serum bilirubin, serum alkaline phosphatase and serum cholesterol were increased, but the partition between cholesterol and esters was normal. In all but one of the instances, result of the cephalin-cholesterol flocculation test was negative. The urine contained large amounts of bile, but urinalysis did not reveal any urobilinogen. The prothrombin time was normal. There was an alteration of the albumin-globulin ratio of the serum in our case, which is in contrast to the normal ratio observed in many of the reported cases of this type of jaundice.

It is very important to be aware of the possible presence of this syndrome, since operation usually is considered to be indicated in cases of jaundice in which these chemical findings are obtained. In cases in which the liver has been damaged, operations are fraught with difficulty because the patients are very poor surgical risks. In cases of jaundice, it, therefore, may be lifesaving to know whether the patients recently have taken chlorpromazine.

In some cases in which jaundice has been caused by the administration of Thorazine, needle biopsy of the liver has disclosed a rather constant picture of stasis in the bile canaliculi and a varying degree of inflammatory infiltration.[8] In these cases, the laboratory findings closely resembled those obtained in seven cases in which jaundice was caused by the administration of methyl testosterone.[12] In the latter cases, the results of the laboratory tests suggested the presence of an obstructive type of jaundice. Similar biochemical findings have been obtained in cases in which jaundice has followed the administration of arsphenamine, thiouracil and other drugs. In discussing the possible cause of the jaundice which followed the administration of methyl testosterone, Werner, Hanger and Kritzler[12] postulated that injury of the hepatic cells by the drug leads to a disturbance of the normal hydration of the bile which becomes too viscous to flow through the intralobular duct system. A similar possibility can be considered in cases in which jaundice has been caused by chlorpromazine.

Why should chlorpromazine injure the hepatic cells? In trying to answer this question, one must consider the following facts. Chlorpromazine is not in itself a hepatotoxic drug.[6] It is impossible to predict the number of cases in which the administration of this drug will cause jaundice. Moyer and his associates[6] said that jaundice has occurred in one of more than 500 cases in which the drug has been administered, while Azima and Ogle[5] said that this complication occurred in 8 of 100 cases. In a case in which a patient has recovered from jaundice induced by this drug, subsequent administration of the drug might not cause jaundice.[3] It appears from reviewing the literature that the drug is most likely to cause jaundice in cases in which it is administered to psychiatric patients. It can be safely assumed that psychiatric patients are not as well nourished as other patients, and this point may be the crux of the problem.

Chlorpromazine is a compound with two methyl groups attached to a nitrogen atom,[13] and it thus may be a biologic antagonist to choline, which has three methyl groups attached to a nitrogen atom. When this drug is administered to poorly nourished patients, it might antagonize the small body stores of choline. When it is administered to well-nourished patients, the stores of choline would overcome this antagonism. Since choline or other compounds with methyl groups in the metabolic pool are necessary for proper functioning of the liver, chlorpromazine might cause jaundice in poorly nourished patients by blocking the small number of methyl groups available. In well-nourished patients, this blocking would not be possible.

This theory would explain why chlorpromazine might not cause jaundice when administered to a patient who has recovered from jaundice induced by the previous administration of this drug. On the second occasion, the patient might be in a better nourished condition. This theory would also explain why chlorpromazine is not a hepatotoxic drug under ordinary circumstances, especially when administered to well-nourished patients.

In the case which we have reported, the most satisfactory rate of recovery occurred

during the period when the patient was given nutritional supplements (table 1). Of course, it is always hazardous to generalize from one case, but the rate of recovery also fits in well with the theory of nutritional antagonism. This theory, if it could be extended to the practical level, would mean that nutritional supplements, including choline, should be given with chlorpromazine unless the patient is obviously well nourished.

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Helpful Signs in the Diagnosis of Functional Disease

Recognition of functional disease is as important as the diagnosis of organic conditions, and perhaps more difficult. History-taking is still most important, but much significant information can also be obtained from a subtle type of physical examination which begins the instant the patient walks into the office. It is hoped that pointing out signs for evaluating functional disease will help the physician base his diagnoses on positive evidence and increase his confidence in making such diagnoses when they are warranted.

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COMMON SENSE AS APPLIED TO RADIATION THERAPY

CRILE[1] recently presented the surgeon's viewpoint of the rational treatment of malignancy. He, along with others, drew an analogy between the metabolism of malignant cells and the various types of bacterial infection. Some malignancies, like infections, are widely disseminated almost from their inception while others remain localized indefinitely. It is obvious that the latter can be eradicated by various means, principally surgical, or by radiation, provided that certain criteria are met. The surgeon must remove all of the neoplasm without cutting through it, or the radiologist must deliver a carcinocidal dosage of radiation to all of the neoplasm.

Radiologists, like surgeons, must remember that they face the law of diminishing returns in treating certain neoplasms. For example, Warren and Witham[2] in a series of 162 autopsies in cases of carcinoma of the breast found only eight cases in which there were no metastases, while in each of the remainder an average of five organs or tissues were involved. The sites of metastases were as follows:

Distant lymph nodes	116
Lung	94
Liver	88
Bone	69
Skin	61
Pleura	56
Adrenal	50
Spleen	23
Peritoneum	21

The purpose of this comment is not to discuss the treatment of this neoplasm but to point out that no matter how early or by what method patients are treated, statistically a certain number cannot be cured. The same statement may be made about many other types of malignancies.

Conversely, certain metastatic lesions may regress provided that the primary lesion can be controlled. Levi and Haig[3] reported two cases of chorionepithelioma with pulmonary metastases which regressed following control of only the primary site. This phenomenon has been seen in cases of lymphoma and on rare occasions with other malignant neoplasms.

Coley[4] warned against the use of roentgen therapy in treating benign bone tumors which are accessible to surgery. He cited the undesirable late effects of radiation on overlying soft tissue, adjacent joint structures, and inhibition of growth and cited the possibility of the development of a malignant tumor at the radiation site. He quoted 11 cases from the literature in which this has happened.

Cipollaro and Crawford[5] warned against radiation overdosage for benign skin lesions. They stated that one must never exceed the ultimate total dosage of 1200 r for these diseases. I have seen many cases of multicentric squamous carcinoma following many years

after radiation for benign lesions of the skin, particularly of the dorsa of the hands. The severe intestinal necrosis with fistula formation and ultimate intestinal stenosis following intensive radiation to the retroperitoneal nodes for control of metastases from seminoma is also fairly common.

The tolerance dosage for normal tissues varies widely. It depends chiefly on the rate of delivery. These dosages have recently been under scrutiny and have been found to be considerably less than previously considered safe. Wachowski and Chenault[6] found degenerative changes in cerebral tissue which were not due to vascular damage following relatively small amounts of radiation.

We must not forget that radiation therapy, like all other therapy, should benefit the patient. We must be careful we do not harm him.

ELMER C. ST. JOHN

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6. Wachowski, T. J. and Chenault, H.: Degenerative effects of large doses of roentgen rays on the human brain. Radiology 45:227 (September) 1945.

THE FUTURE OF SURGERY

One of the hit songs of "Oklahoma!" croons:

"Everything's up to date in Kansas City
They've gone about as far as they can go."

Frequently since about 1880 medical philosophers have been saying the same about surgery. Winston Churchill has suggested that those men can look furthest forward who can look furthest back. In a new discussion of the future of surgery, Sir Heneage Ogilvie[1] lists some of the great advances by which surgery has advanced in recent years:

(1) Continuous administration of fluids introduced by Hendon and Matas in 1926; (2) continuous gastric suction suggested by John Ryle in 1920 and advocation of the principle of intestinal decompression by Wangensteen in 1932; (3) blood transfusion begun by Lewisohn in New York and perfected by Marriott and Kekwick in 1935; (4) more recently advances in surgery are related to problems of fluid and electrolyte balance and Ogilvie assigns much of the credit to Frances Moore of the Peter Bent Brigham Hospital of Boston.

Add to the discoveries already listed the advances in chemotherapy, hemostasis by electrocoagulation, new surgical materials for repairs and prostheses, and modern anesthetics—these make for safety in surgery beyond anything that any surgeon dreamed previously. Here is a picture of the modern anesthetist in action:

> The modern anaesthetist gains the patient's confidence by a complete pre-operative overhaul, prepares him by a careful assessment and adjustment of his deficiencies, allays his fears by pre-operative medication, renders him unconscious by an intravenous barbiturate, maintains analgesia by one of the anaesthetic gases, ensures oxygenation and immobility of the diaphragm by controlled respiration, and by the use of relaxants provides the degree of muscular relaxation needed at any stage of the operation. He is no longer a mere "dope merchant." He takes over the whole responsibility for the patient's welfare and the surgeon's peace of mind. He presents the operator with tissues in a condition to suit him, relieves him of all anxiety except for the technical details of the operation itself, and hands him back a patient alive and well and about to come around.

For the future Ogilvie sees a succession of new antibiotics, analgesics, sedatives and ganglion blockers. He hopes for a contrast medium excreted by the pancreas to provide an estimate of pancreatic function. He desires a systemic hemostatic that will arrest bleeding from small vessels cut at operation. Departing into the realm of imagination, Sir Heneage sees a surgical approach to the personality or the spirit of man by procedures related to the glands and the brain. "The anxious restless-

ness of thyrotoxicosis, the lethargy of myxedema, the aggressiveness of adolescence and the unambitious placidity of the eunuch, the sudden rages of the adrenal hypertensive, the bewildering loneliness of the hermaphrodite—these are familiar conditions which offer a challenge to the surgeon who is a physiologist."

Few physicians have contemplated fully the experimentation now in progress with surgical methods that affect mentality. Here Ogilvie suggests:

> Hemispherectomy, while it cannot restore the paralysis of a spastic child, will make it no worse, and will put an end to the fits and the rages that made it ineducable. Identification and removal of a trigger focus in the temporal lobe may transform a handicapped epileptic into a useful citizen. Operations on the frontal lobes may enable a manic patient to leave hospital and fill a useful place in society.
>
> These operations, apart from thyroidectomy for thyrotoxicosis, are recent developments, the first incursion of surgery into the field of pneumatic therapy. Much research is being undertaken into the function of the cortical areas and subcortical pathways of the frontal lobes in the endeavour to plan operations that can relieve tension without any associated deterioration in character.

With such a future challenging the young surgeon, the opportunity for those who are qualified to lead is tremendous. Manual skill has not changed greatly in some centuries although systematic training has contributed vastly to its development. In addition to manual skill Ogilvie sees the need for eight other qualities in his degree. The great surgeon must have:

> . . . knowledge of the basic sciences, and particularly of pathology, of the theory and practice of clinical and operative surgery, and of the writings of other surgeons. He must have wisdom, the blending of knowledge and experience into a personal outlook that will guide him to the solution of fresh problems. He must have the power of communicating his views to others, of clear writing, and clear speaking, which in turn arise from clear thinking. He must have originality, the ability to find in his experience the path that leads to fresh knowledge and enables him to leave surgery in some way better for his stewardship. He must have leadership, the power to convince contemporaries and to fire juniors with his own enthusiasm. He must have integrity, absolute standards of honesty, justice, and humanity. He must have courage to hold on in difficulty, to persevere in failure, to back what he believes to be right though all men attack him. He must also have humility, a consciousness of the great disparity between his achievements and his hopes.

Turning his attention specifically to Great Britain, Sir Heneage Ogilvie paints a rather dismal picture. The processes of selection that led to the development of great surgeons in the past have disappeared in the regimentation of the Welfare State.

> The contributions of Germany to surgical advance in the last 50 years have been pathetic.
>
> Our British traditions have now been cast aside. In the place of surgery as a vocation, a dangerous adventure calling to the stout of heart, we have surgery as a guaranteed career, offering a fixed number of beds requiring a fixed number of sessions to look after them, a steady supply of patients, a scheduled income rising steadily with seniority rather than proficiency, little chance of promotion however good one's work may be, employment till a statutory age, however one may deteriorate, no opportunity of further employment after that age however good one may be at the time, and a pension on retirement. To this safe haven, offered by the Welfare State, many are called, but few are chosen.

Dr. Ogilvie, who is consulting surgeon to Guy's Hospital and the Royal Masonic Hospital, feels that the maintenance of private practice in some form is essential not merely to the future advance of British surgery but to the integrity of the National Health Service. Here is a pungent contrast:

> The danger of the present position, as I see it, lies in the substitution of a label for reality, of an awarded position for an earned position. Formerly a surgeon was what he made himself in the eyes of his fellows. He started as one to be watched, became one to be employed, and finally one to be consulted. To-day he walks into a board-room a registrar and comes out a consultant. Formerly he

climbed the ladder of professional standing carefully, under watchful and not always friendly eyes. He studied what had already been done and what results had already been obtained before he started to invent new methods, and he made very sure of the soundness of his innovations before he published them. To-day he is no sooner appointed than he designs a new operation, one that ignores past experience and physiological principles, and he blares it abroad with all the ballyhoo of an advertisement for a new detergent without waiting to observe its results. It is as wrong for a surgeon to undertake a difficult operation for which he has not been trained as it is for a man to run a sports car through a football crowd before he has passed his driving test. It is as criminal for a surgeon to practise and publish a new operation without complete study as it is for a manufacturer to put a dangerous drug on the market without previous laboratory tests.

How fortunate we are here in the United States to have resisted thus far the stagnation and the oppression that come from the regimentation imposed by the weight of the dead hand of the State. American medicine is experimenting with full-time salaried surgeons, full-time professors, part-time teachers who have private practice—indeed all the variations to which the situation is susceptible. The danger lies not in experimentation and evolution but in failure to recognize the fundamental considerations so effectively set forth by Sir Heneage Ogilvie.

MORRIS FISHBEIN

1. Ogilvie, Sir Heneage: The future of surgery. Brit. M. J. 2:1435-1439 (December 18) 1954.

EDUCATION IN NUTRITION

The average man in the United States today, according to Dr. William H. Sebrell, Jr.,[1] of the U. S. Public Health Service, obtains enough food to prevent serious deficiency diseases. This does not mean, however, that he is obtaining enough food of the right kind to give him optimal health. Neither does it mean that his children have the proper food for optimal growth, for developing resistance to disease, for preventing chronic disease, for overcoming unusual stress on the human body which may be associated with infection or serious illness, or to give them sufficient extra energy to enable them to keep pace with current drives.

The answer to the problem of improving nutrition is probably to be found in better education in the field of nutrition. A survey made by General Mills, Inc. covering 60,000 children in 38 states indicates that children's diets become poorer as the children grow older, that boys' diets are notably deficient in fruits and vegetables, that diets are related closely to the occupation and economic status of the parents, and that adolescent girls drink far too little milk in the belief that milk is especially fattening. Particularly significant in the field of diet is the choice of foods for pregnant women and industrial workers and for the aging population. No doubt nutritional science can do about as much for meeting the problems of the aged as any other form of scientific control.

In the United States the choice of food for the family is usually made by the mother in her visits to the market. Dr. Sebrell feels that she does not get proper guidance at the point of sale to help her make the purchases follow what she has been taught about nutrition. As she walks through the market deciding what she will buy, she gets adequate information concerning prices, sizes, weights, quality and other factors, but the labels do not indicate nutritional values, and educational material on nutritional values is sadly lacking. The American diet is deficient, if anything, in vitamin C, calcium and vitamin A.

Dr. Sebrell's final conclusion is: "We will never have a really successful program until the food sellers and the food industry cooperate with the scientists, nutritionists and teachers in developing a positive, aggressive, co-ordinated program to present basic nutrition facts clearly and simply to the American housewife." Here is a challenge that enterprising purveyors of food can readily meet and thus render a real public service.

MORRIS FISHBEIN

1. Sebrell, William H., Jr.: Nutrition at the shopping center. Pub. Health Rep. 70:561 (June) 1955.

Program

40th Interstate Postgraduate Medical Assembly

Milwaukee, Wisconsin — November 14-17, 1955

Program Schedule

Monday, November 14, 8:25 A.M.

8:25 Welcome.

Dr. David V. Elconin, President, The Medical Society of Milwaukee County.

8:30 Clinic: Newer Treatments for Pernicious Anemia.

Dr. John S. Hirschboeck, Dean and Associate in Medicine, Marquette University School of Medicine, Milwaukee.

9:00 Clinic: Clinical Discussions of Neurology and Neurologic Surgery.

Dr. Paul C. Bucy, Professor of Surgery, Northwestern University School of Medicine; Director of Division of Neurologic Surgery, Wesley Memorial Hospital, Chicago.

9:30 Address: Mechanical Treatment of Pulmonary Failure.

Dr. John E. Affeldt, Instructor of Medicine, College of Medical Evangelists School of Medicine, Los Angeles; Chief Physician, Respiratory Center for Poliomyelitis, Rancho Los Amigos, Hondo, California.

10:00-11:00 A.M. *Intermission for Review of Exhibits*

11:00 Address: Clinical Considerations of Ovarian Cystomas.

Dr. Clyde L. Randall, Professor of Obstetrics and Gynecology, University of Buffalo School of Medicine, Buffalo, New York.

11:30 Address: Regional Enteritis—Diagnosis and Medical Management.

Dr. William G. Sauer, Consultant in Medicine, Mayo Clinic; Assistant Professor of Medicine, Mayo Foundation, University of Minnesota Graduate School, Rochester, Minnesota.

Noon Intermission

1:30 P.M.

1:30 Clinic: Strictures of the Common Duct.

Dr. Warren H. Cole, Professor of Surgery and Head of the Department, University of Illinois College of Medicine, Chicago.

2:00 Address: Helpful Signs in the Recognition of Functional Disease.

Dr. Leonard L. Lovshin, Department of Medicine, Cleveland Clinic, Cleveland.

2:30 Address: The Care of the Seriously Ill Asthmatic Patient.

Dr. Haddon M. Carryer, Mayo Clinic; Assistant Professor of Medicine, Mayo Foundation, University of Minnesota Graduate School, Rochester, Minnesota.

3:00-4:00 P.M. *Intermission for Review of Exhibits*

4:00 Address: A New Method of Treating Emotionally Induced Illness Adaptable to General Practice.

Dr. John A. Schindler, Department of Internal Medicine, The Monroe Clinic, Monroe, Wisconsin.

4:30 Clinic: Fractures in Children.

Dr. Walter P. Blount, Head of the Orthopaedic Department, Milwaukee Children's Hospital, Milwaukee.

5:00 Address: Hypogonadism in Middle-Aged and Elderly Men.

Dr. John Esben Kirk, Associate Professor of Gerontology, Washington University School of Medicine, St. Louis.

Evening Program

7:30 P.M.

7:30 (Speaker to be announced)

8:00-9:00 Panel: Ulcers and Cancer of the Stomach.

Dr. Warren H. Cole, Professor of Surgery and Head of the Department, University of Illinois College of Medicine, Chicago, Moderator.

Dr. Walter L. Palmer, Richard T. Crane Professor of Medicine, University of Chicago School of Medicine, Chicago.

Dr. Warren W. Furey, Clinical Professor of Roentgenology, Stritch School of Medicine, Loyola University, Chicago.

9:00 Questions with Moderator.

Tuesday, November 15, 8:00 A.M.

8:00 Address: Evaluation of the Methods of Treatment of Essential Hypertension.

Dr. Francis D. Murphy, Professor of Medicine and Head of the Department, Marquette University School of Medicine; Medical Director, Milwaukee County Hospital, Milwaukee.

8:30 Clinic: The Management of Rheumatoid Arthritis.

Dr. Charles Ragan, Associate Professor of Medicine, Columbia University College of Physicians and Surgeons, New York.

9:00 Clinic: Present Day Concepts of Muscular Dystrophy and Atrophy.

Dr. Hans H. Reese, Professor of Neuropsychiatry, University of Wisconsin Medical School, Madison.

9:30 Address: Newer Concepts in the Treatment of Cleft Palate and Harelip.

Dr. Wayne B. Slaughter, Professor of Surgery, Stritch School of Medicine, Loyola University, Chicago.

10:00-11:00 *Intermission for Review of Exhibits*

11:00 Clinic: Carcinoma of the Thyroid.

Dr. Richard B. Cattell, Director, Lahey Clinic; Surgeon-in-Chief, New England Baptist Hospital; Surgeon, New England Deaconess Hospital, Boston.

11:30 Address: Tumors in Infancy and Childhood.

Dr. Tague C. Chisholm, Clinical Assistant Professor of Surgery, University of Minnesota Medical School, Minneapolis.

Noon Intermission

1:30 P.M.

1:30 Clinic: Intra-articular Steroids: Clinical Applications and Laboratory Effects.

Dr. Ivan F. Duff, Associate Professor of Medicine, University of Michigan Medical School, Ann Arbor, Michigan.

2:00 Address: Operative Treatment of Uterine Prolapse.

Dr. Ralph A. Reis, Professor of Obstetrics and Gynecology, Northwestern University School of Medicine, Chicago.

2:30 Address: Jaundice.

Dr. Leon Schiff, Professor of Clinical Medicine, University of Cincinnati College of Medicine; Director, Gastric Laboratory, Cincinnati General Hospital, Cincinnati.

3:00-4:00 *Intermission for Review of Exhibits*

4:00-5:00 Panel: Multiple Sclerosis.

Dr. Pearce Bailey, Clinical Professor of Neurology, Georgetown University School of Medicine, Washington, D. C.; Director, National Institute of Neurological Diseases and Blindness of the Public Health Service, Bethesda, Maryland, Moderator.

Dr. Roland P. Mackay, Professor of Neurology, University of Illinois College of Medicine, Chicago.

Dr. Francis J. Millen, Assistant Professor of Neurology, Marquette University School of Medicine, Milwaukee.

5:00 Clinic: Intestinal Obstruction.

Dr. Rudolf J. Noer, Professor and Chairman of the Department of Surgery, University of Louisville School of Medicine, Louisville, Kentucky.

Evening Program

7:30 P.M.

7:30 Address: Recent Advances in the Diagnosis of Virus Diseases, with Special Reference to Tissue Cultures.

Dr. Andrew J. Rhodes, Professor of Virus Infections, The School of Hygiene, University of Toronto; Director, the Research Institute, the Hospital for Sick Children, Toronto, Ontario.

8:00 Address: The Use and Abuse of Sedative Drugs.

Dr. Franklin G. Ebaugh, Clinical Professor of Psychiatry, University of Colorado Medical School, Denver, Colorado.

8:30-9:30 Panel: Diverticulitis and Diverticulosis.

Dr. Richard B. Cattell, Director, Lahey Clinic; Surgeon-in-Chief, New England Baptist Hospital, Boston, Moderator.

Dr. Leon Schiff, Professor of Clinical Medicine, University of Cincinnati College of Medicine; Director, Gastric Laboratory, Cincinnati General Hospital, Cincinnati.

Dr. S. Archibald Morton, Professor and Director of the Department of Radiology, Marquette University School of Medicine, Milwaukee.

Wednesday, November 16, 8:00 A.M.

8:00 Address: Endocrine Therapy in Gynecology.

Dr. Willard M. Allen, Professor of Obstetrics and Gynecology and Head of the Department, Washington University School of Medicine, St. Louis.

8:30 Clinic: Both-Bone Fractures of the Leg.

Dr. Carlo Scuderi, Associate Professor of Surgery, University of Illinois College of Medicine; Senior Attending Surgeon, Cook County Hospital, Chicago.

9:00 Clinic: The Papulosquamous Skin Conditions, Their Diagnosis and Treatment.

Dr. Sture A. M. Johnson, Professor of Dermatology and Syphilology, University of Wisconsin Medical School, Madison.

9:30 Address: Recent Significant Developments in Intracardiac Surgery.

Dr. Charles P. Bailey, Professor and Head of Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia.

10:00-11:00 *Intermission for Review of Exhibits*

11:00 Address: A Current Evaluation of the Antibiotics in Surgery.

Dr. William A. Altemeier, Christian R. Holmes Professor of Surgery and Director of the Department, University of Cincinnati College of Medicine, Cincinnati.

11:30 Clinic: Fracture Dislocations of the Thoracolumbar Spine with Spinal Cord Injury.

Dr. E. Harry Botterell, Associate Professor of Surgery (Neurosurgery), University of Toronto, Toronto, Ontario.

Noon Intermission

1:30 P.M.

1:30 Address: Retrolental Fibroplasia. The Schneider Foundation Eye Presentation.

Dr. John C. Locke, Demonstrator, Department of Ophthalmology, McGill University, Montreal, Quebec.

2:00-3:00 Symposium: Trauma.

Dr. Richard W. Zollinger, Assistant Clinical Professor of Surgery, Ohio State University College of Medicine, Columbus, Ohio, Moderator.

Dr. George J. Curry, Chief, Section for the Surgery of Trauma, Hurley Hospital, Flint, Michigan; Chairman, Committee on Transportation of the Injured, American College of Surgeons.

Dr. E. Harry Botterell, Associate Professor of Surgery (Neurosurgery), University of Toronto, Toronto, Ontario.

3:00-4:00 *Intermission for Review of Exhibits*

4:00 Clinic: Surgery of Diseases of the Lung.

Dr. Alton Ochsner, The William Henderson Professor of Surgery and Chairman of the Department of Surgery, Tulane University School of Medicine; Director of Surgery, Ochsner Clinic and Ochsner Foundation Hospital, New Orleans.

4:30 Address: Problems of Ovarian Tumors.

Dr. Fred J. Hofmeister, Assistant Clinical Professor of Obstetrics and Gynecology, Marquette University School of Medicine, Milwaukee.

5:00 (Speaker to be announced)

Evening Program

DINNER AND ENTERTAINMENT

EMPIRE ROOM, HOTEL SCHROEDER

6:30 P.M.

Presiding: **Dr. Edwin S. Hamilton,** Chairman, Board of Trustees, Interstate Postgraduate Medical Association.

Speaker: **Dr. Charles W. Mayo,** Incoming President: The Role of Medicine and Doctors in International Affairs.

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Thursday, November 17, 8:00 A.M.

8:00 Address: Newer Concepts of the Epidemiology of Sarcoidosis (A Study Based on Experience in 172 VA Hospitals).

Dr. Martin M. Cummings, Director of Research, Veterans Administration, Washington, D. C.

8:30 Address: Fetal Resuscitation and Respiration.

Dr. Virginia Apgar, Professor of Anesthesiology, Columbia University College of Physicians and Surgeons, New York.

9:00 Clinic: Surgical Lesions of the Adrenal Gland.

Dr. Waltman Walters, Mayo Clinic; Professor of Surgery, University of Minnesota Graduate School of Medicine, Rochester, Minnesota.

9:30 Address: Malignant Hypertension.

Dr. James A. Evans, Department of Internal Medicine, Lahey Clinic, Boston.

10:00-11:00 *Intermission for Review of Exhibits*

11:00 Address: The Present Status of Surgery in the Treatment of Cancer.

Dr. George Crile, Jr., Cleveland Clinic, Cleveland.

11:30 Clinic: The Treatment of Bulbar Poliomyelitis.

Dr. Martin H. Seifert, Associate Professor of Medicine, Northwestern University School of Medicine, Chicago.

Noon Intermission

1:00 P.M.

1:00 Annual Business Meeting and Presentation of Special Attendance Awards to Members.

1:30 Address: The Diagnostic Problem of Histoplasmosis.

Dr. John J. Procknow, Assistant Professor of Medicine, Section of Preventive Medicine, Department of Medicine, University of Chicago School of Medicine, Chicago.

2:00 Address: The Late Treatment of Burns.

Dr. Clifford L. Kiehn, Professor of Plastic Surgery, Western Reserve University School of Medicine, Cleveland.

2:30-3:00 *Intermission for Review of Exhibits*

3:00 Address: Why Not "Cosmetic" Transfusions?

Dr. Tibor J. Greenwalt, Medical Director, Milwaukee Blood Center; Assistant Clinical Professor of Medicine, Marquette University School of Medicine, Milwaukee.

3:30 Clinic: Cancer of the Mouth and Neck.

Dr. H. Mason Morfit, Associate Professor of Surgery and Director of the Bonfils Tumor Clinic, Colorado University School of Medicine, Denver.

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THE MEDICAL *Bookman*

► A PRIMER OF ELECTROCARDIOGRAPHY

By George E. Burch, M.D., Henderson Professor of Medicine, Tulane University School of Medicine, New Orleans, and Travis Winsor, M.D., Assistant Clinical Professor of Medicine, University of Southern California Medical School, Los Angeles. Ed. 3. 286 pages with 281 illustrations. 1955, Lea & Febiger, Philadelphia. $5.00.

The third edition of Burch and Winsor's "Primer," like the second, is a good introduction to this field of study. The fundamental nomenclature is outlined in the first chapter, as it should be in an elementary text of this sort. The chapter titles and subheadings are clear and concise. The material is, by and large, adequate. As the authors state, the text is dogmatic, even to the illustrations, which do not prepare the student for the varied forms of recordings he will meet in the clinical electrocardiogram within each disease state. The chapter dealing with disorders of the heartbeat and the appendix containing the tables of normal values probably will continue to be consulted by the student for some time after he has passed the stage at which a primer can be useful to him.

There has been no fundamental change in the organization of the new edition. Most notable among the revisions are the addition of a chapter on the spatial vectorcardiogram and an expansion of the section dealing with unipolar leads and precordial leads. It is disappointing that, although the unipolar leads and precordial leads have been given expanded space, the explanations of disease states and examples of electrocardiographic patterns still are given almost exclusively in terms of the three standard leads.

J. A. C.

► BICKHAM-CALLANDER SURGERY OF THE ALIMENTARY TRACT

By Richard T. Shackelford, M.D., Assistant Professor of Surgery, assisted by Hammond J. Dugan, M.D., Assistant in Surgery, The Johns Hopkins University School of Medicine, Baltimore. Vols. 1, 2 and 3. 2,575 pages with 1,705 illustrations. 1955, W. B. Saunders Company, Philadelphia and London. $60.00.

Bickham's "Surgery of the Alimentary Tract" has been completely rewritten by Dr. Richard T. Shackelford. The task was undertaken by the late Dr. C. Latimer Callander, but at his death the text was not in sufficiently finished form for publication. Dr. Shackelford, assisted by Dr. Hammond J. Dugan, has since finished the revision, which is now published in three volumes.

The author has covered surgery of the alimentary tract comprehensively and has made the book almost an encyclopedia of the surgical procedures that have been proposed for treating surgical lesions of this system of the body. In addition, he has evaluated these procedures in the light of his experience.

The text is well organized. Much of the material and many illustrations have been borrowed from original sources, which makes the book an excellent reference for contributions to this field of surgery. Related physiology and anatomy are well covered. Volume I deals with the esophagus, stomach and duodenum, liver and biliary tract. Volume II covers the pancreas, small intestine and colon. Volume III covers the anorectal canal, hernias of the gastrointestinal tract and, together with related anatomy, incisions for approaching the various segments of the alimentary canal.

O. H. B.

(Continued on page A-102)

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Interstate Postgraduate Medical Association Assembly, Milwaukee, November 14 to 17, 1955

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► KINESIOLOGY OF THE HUMAN BODY UNDER NORMAL AND PATHOLOGICAL CONDITIONS

By Arthur Steindler, M.D., Emeritus Professor of Orthopedic Surgery, State University of Iowa, Iowa City. 708 pages, illustrated. 1955, Charles C Thomas, Springfield, Illinois. $19.75.

The presentation of a subject requiring mathematical calculation for a specific definition has an uncertain prognosis for acceptance by the reading audience. It immediately will alienate a certain few who feel strained by what they consider unnecessary concentration. However, the factual approach to the subject of kinesiology must in essence deal with mechanical, and hence mathematical, consideration. This feature is inherent in the descriptive evaluation of normal and pathologic processes of locomotion. The author recognizes that this detail adds a laborious characteristic and has included only the barest minimum. Sketches, x-ray prints and photographs have been included wherever necessary for better definition.

The book is unusually practical, especially in the realms of orthopedic surgery, physical medicine and rehabilitation. The value is not limited to those fields, however, and there is useful content for persons interested in education, functional physiology and disability evaluations.

The subject matter is comprehensive and has a thesis for advanced study. The vast amount of applicable clinical information included will please the practicing physician.

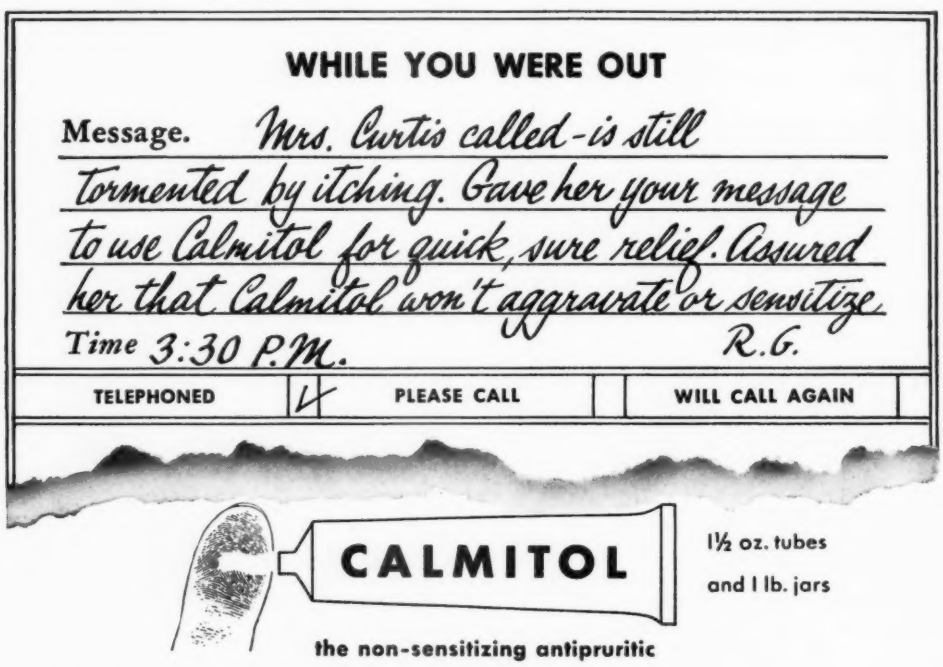
H. O'P.

► MEDICAL AND PUBLIC HEALTH LABORATORY METHODS

Edited by the late James Stevens Simmons, M.D., Dean and Professor of Public Health, Harvard School of Public Health, Boston, and Cleon J. Gentzkow, M.D., Director, Bureau of Laboratories, Department of Health, Commonwealth of Pennsylvania, Philadelphia. Ed. 6. 1,191 pages, illustrated. 1955, Lea & Febiger, Philadelphia. $18.50.

The previous edition of this book, published under the title, "Laboratory Methods of the United States Army, Ed. 5," was prepared to

(Continued on page A-105)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meet a wartime need during World War II. This new edition, with a more appropriate name, covers all of the basic information of a military manual with much additional information which has been made available in the past 11 years.

The book is arranged in 11 major parts with 52 chapters prepared by 36 authors; of these, 24 are new in this edition. The entire book has been expanded and includes more illustrations than the previous edition. The first two parts, on clinical pathology and chemistry, have been increased by more than 200 pages. In the section dealing with bacteriology, a short new chapter on antibiotics has been added.

As in the past, this book will continue to be useful to personnel in both hospital and office diagnostic laboratories, as well as to those who deal with military and public health problems.

A. H. S.

► THE LUNG; Clinical Physiology and Pulmonary Function Tests

By Julius H. Comroe, Jr., M.D., Robert E. Forster, II, M.D., Arthur B. Dubois, M.D., William A. Briscoe, M.D. and Elizabeth Carlsen, A.B., Department of Physiology, University of Pennsylvania Graduate School of Medicine, Philadelphia. 219 pages with 57 illustrations. 1955, The Year Book Publishers, Inc., Chicago. $5.50.

This concise monograph of the current concepts of respiratory physiology and practical application of the measurement of pulmonary function is divided into three parts. Part 1 presents the principles of pulmonary physiology as applied to man in health and disease. Excellent diagrams clearly illustrate the basic aspects of respiration and the close relationship of these integral functions.

Part 2 includes a series of clinical cases illustrating the wide variety of application of the basic principles set forth in Part 1.

Part 3 is a comprehensive appendix containing normal values and many established equations and calculations used in the investigation of both the basic and practical aspects of pulmonary function. The authors did not attempt to describe the technical details of performing the many tests of cardiopulmonary function.

Parts 1 and 2 are required reading for practitioners interested in pulmonary diseases. The

(Continued on page A-106)

entire book will serve as a ready reference for those clinicians who teach in the field of pulmonary diseases.

R. D. M.

► CARDIOLOGY NOTEBOOK

Edited by Alfred P. Fishman, M.D., Columbia University College of Physicians and Surgeons, New York, with five co-editors. 97 pages, illustrated. 1955, Grune & Stratton, Inc., New York and London. $2.50.

This volume was designed to fill a special need in the undergraduate curriculum of a major medical school. The idea was "to make readily available to him [the medical student], in the simplest form, a few basic examples of the method and language of cardiology. . . ." To do this, the authors have supplemented the basic technic of bedside clinical evaluation by presenting a review of the principal laboratory procedures utilized by the cardiologist.

The first of four sections is an adequate introduction to cardiac fluoroscopy, with emphasis on the correlation of anatomic structures and x-ray shadows in the normal person and in certain common abnormal conditions. The second section presents a selected group of electrocardiograms (without clinical data) illustrating the common arrhythmias, disturbances in conduction and patterns in infarction. Unfortunately, there is considerable loss of detail in the reproduction of several of the tracings. Some criticism could be directed to the interpretation of many of the tracings. Such comments as "The T waves, although obscure at times, suggest the presence of myocardial disease," "No axis deviation" and "No definite atrial activity can be seen" are insufficiently accurate and may lead the student to certain misconceptions.

A section on hemodynamics tabulates average normal values for such physiologic variables as arterial oxygen and carbon dioxide content, cardiac output, blood volume, vital capacity and respiratory quotient. No comment is made on the clinical application of such determinations. Examples of data obtainable by direct arterial puncture and by venous catheterization of the heart are included. A synopsis of nomenclature for cardiac diagnosis and an index complete the volume.

J. C. B.

(Continued on page A-108)

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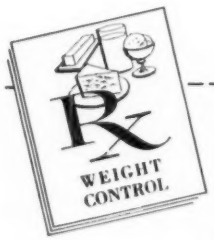

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► GYNAECOLOGY

By Douglas H. MacLeod, M.S., Gynaecological Surgeon, St. Mary's Hospital, and Charles D. Read, M.B., Director, Institute of Obstetrics and Gynaecology, British Postgraduate Medical Federation, University of London, London. Ed. 5. 864 pages with 551 illustrations including 27 in color. 1955, Little, Brown and Company, Boston. $16.00.

This modernized version of the well-known gynecologic classic, published originally by Eden and Lockyer, has been cited as the most complete single volume work on gynecology in the English language. To each section is appended a carefully chosen selection of references which, if read, should greatly broaden the reader's knowledge. The importance of the lymphatic drainage and the innervation of the pelvic organs is stressed in a new chapter on anatomy. Well outlined are disorders of function and development of the genital system as effected by the various abnormalities of the endocrine glands.

The intimation that surgical intervention should be chosen in preference to x-ray and radium therapy as primary treatment for carcinoma of the cervix, grades 1 and 2, is questionable. This is the only point on which constructive criticism might be offered.

Because of its broad coverage of the subject of gynecology, the book would seem to serve best as an introduction to gynecology for the medical student or as an excellent review for the physician in general practice.

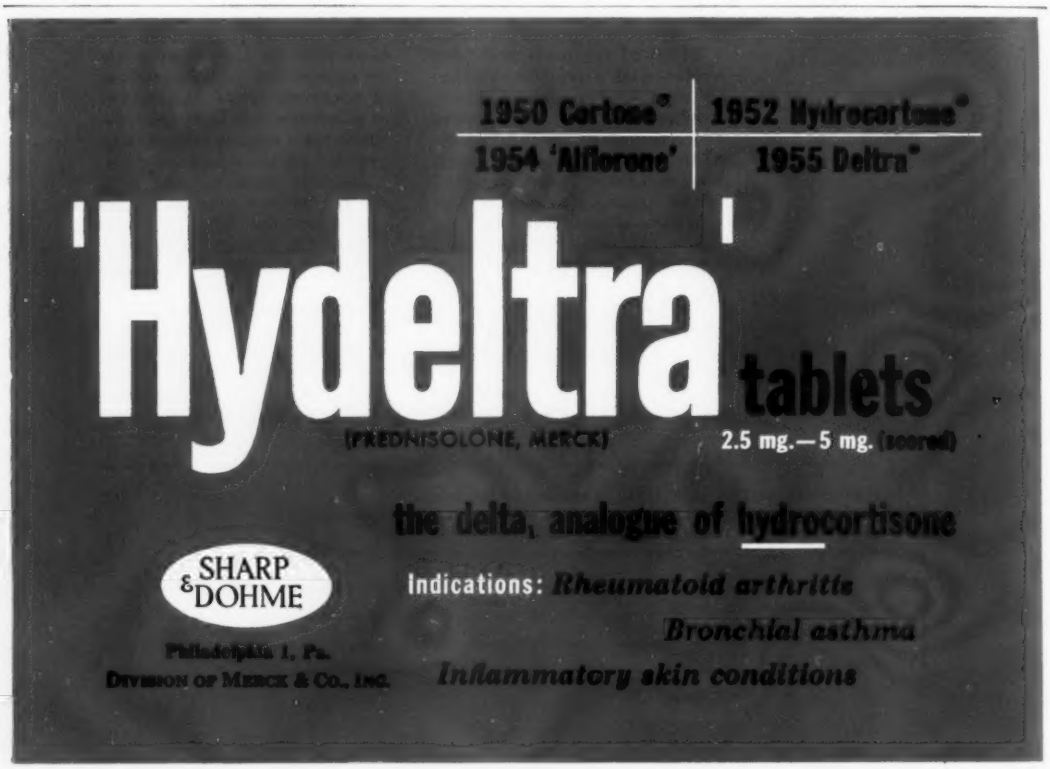
R. A. S.

► PAIN: ITS MECHANISMS AND NEUROSURGICAL CONTROL

By James C. White, M.D., Associate Professor of Surgery, and William H. Sweet, M.D., Associate Clinical Professor of Surgery, Harvard Medical School, Boston. 736 pages with 134 illustrations and 53 tables. 1955, Charles C Thomas, Springfield, Illinois. $17.50.

In the midst of the constant demands on the slender resources of the usual medical student for books of all kinds on the basic medical sciences, the expenditure of $17.50 for a single volume on pain may seem fanciful indeed. Yet, if

(Continued on page A-112)



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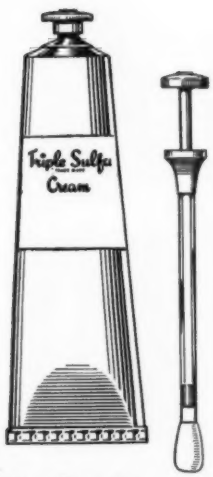

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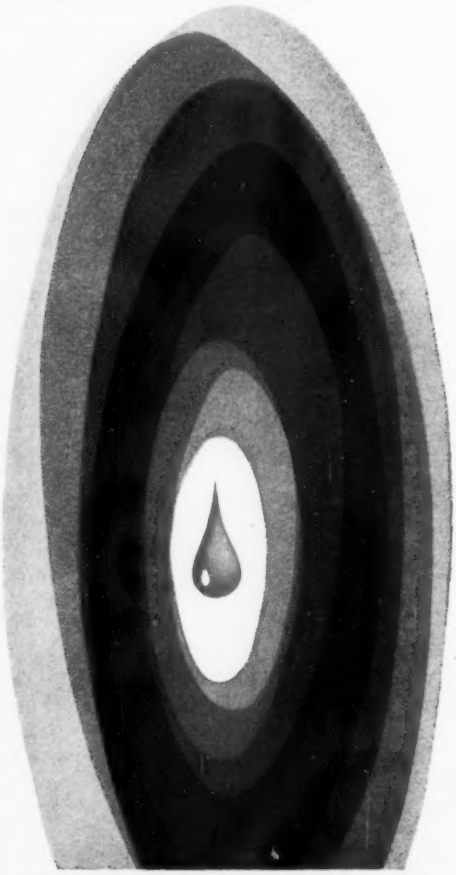

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the student will but reflect, he will come inevitably to the realization that virtually every problem of treatment which he will ever encounter during his career as a physician will be characterized by either pain or discomfort.

Viewed from such an aspect, the broad experience of two recognized algesiologists such as Professors White and Sweet can be of infinite value to the student who needs much information on a variegated subject compressed into one volume. The main basis for this volume is the experience of the authors in treating 420 patients with severe, persistent pain from 1935 to 1949, inclusive. Some of the material in the work, as is commonly the case, has appeared in medical journals, but not in such an integrated background as a full-length volume provides.

Since the experience reflected in the volume is not open to caveat, the next most valuable feature is the arrangement of the text, so far as a busy student is concerned. From the basic physiologic and anatomic features of the structures concerned in pain, the text proceeds to psychiatric considerations, to surgical corrective technics and finally to the treatment of specific pain entities. It is the orderly progression of material in logical sequence which will conserve the time of the student who uses this book, and possibly impart to him at least some of the equanimity which Sir William Osler thought he needed.

J. S. L.

Books received will be acknowledged in this department each month. As space permits, books of principal interest to our readers will be reviewed more extensively.

The Practice of Dynamic Psychiatry. By Jules H. Masserman, M.D., Professor of Neurology and Psychiatry, Northwestern University, Chicago. 790 pages. 1955, W. B. Saunders Company, Philadelphia and London. $12.00.

Transplantation of Tissues. By Lyndon A. Peer, M.D., Clinical and Research Director, Rehabilitation Center for Plastic and Reconstructive Surgery, St. Barnabas Hospital, Newark, New Jersey. Vol. 1. 421 pages with 163 illustrations. 1955, The Williams & Wilkins Co., Baltimore. $13.50.

It All Started with Europa. By Richard Armour, Ph.D. 119 pages. 1955, McGraw-Hill Book Company, Inc., New York. $2.75.

The Therapy of Skin Tuberculosis. By Gustav Riehl, M.D., Professor of Dermatology, University of Vienna, and Oswald Köpf, M.D., Wilhelminen Hospital, Vienna. 247 pages with 8 illustrations. 1955, Charles C Thomas, Springfield, Illinois. $6.75.